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DISEASES
OF THE INTESTINES

BY

DR. I. BOAS

SPECIALIST FOR GASTRO-INTESTINAL DISEASES IN BERLIN

AUTHORIZED TRANSLATION FROM THE FIRST GERMAN EDITION
WITH SPECIAL ADDITIONS

BY

SEYMOUR BASCH, M. D.

NEW YORK CITY

"Nec ultra, nec infra scire"

WITH FORTY-SEVEN ILLUSTRATIONS

NEW YORK
D. APPLETON AND COMPANY
1902

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1902

AUTHOR'S PREFACE TO THE AMERICAN TRANSLATION

It affords me great pleasure to make a few introductory remarks to this translation of my recently published *Diagnostik und Therapie der Darmkrankheiten.*

When the present book was written, the interest of the profession in the pathology of the alimentary tract had already become very great. Owing to the ease with which diseases of the œsophagus and stomach could be investigated, these had been extensively studied. Because of their inaccessibility and the difficulty of judging the effect of treatment, our knowledge of affections of the intestine (exclusive, perhaps, of the rectum) was still very meagre.

Notwithstanding that the classical publications of Woodward and Nothnagel, and the studies in ptomainology and the pathology of metabolism have done much to further our knowledge of intestinal diseases, we have advanced but little since the days of Henoch, Bamberger, von Leube, and their contemporaries.

Internal medicine must acknowledge a debt of gratitude to surgery, for the surgeon has contributed most to the progress that has been made. Our knowledge of appendicitis, intestinal obstruction and stenosis, and benign and malignant tumours has been greatly enriched by the results obtained from surgical treatment, and this progress is still going on. It is universally conceded that the American profession has contributed much toward this end, and in many parts of this work I have acknowledged this indebtedness. Numerous references to American authors will be found throughout the book.

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It might be inferred from this that my treatise contains little that is unknown in America, but I trust that the American reader will find some useful diagnostic and therapeutic hints in the following pages.

I desire to express my thanks to my former assistant, Dr. Basch, for having undertaken and carried to a successful conclusion the work of translation.

I trust that the American edition will meet with the same success that has attended the original in Germany. If it will aid the practitioner in solving some of the difficult problems in intestinal pathology and assist him in the treatment of his patients, the author will feel that his labours have not been in vain.

I. Boas.

Berlin, *February, 1901.*

TRANSLATOR'S PREFACE

The popularity which Dr. Boas's treatise has enjoyed abroad, and the absence in the English language of any detailed and exhaustive work on intestinal diseases, have led to the publication of the present translation.

The book is intended more especially for the requirements of the general practitioner, but on account of the exhaustive and concise description of physiologico-chemical processes and laboratory methods, it must also prove of value to other scientific investigators.

Additions have been made to the chapters on Appendicitis and Hydrotherapeutics, a special account given of the intestinal gases, and brief notes added in various parts of the book. These are indicated by [ ].

I wish to express my sincere thanks to Dr. S. Neuhof, of this city, for valuable assistance in the preparation of this work for the press, and to the publishers, D. Appleton and Company, for the many courtesies extended to me.

S. Basch.

48 East Sixty-third Street,
New York, *March, 1901.*

PREFACE TO THE FIRST GERMAN EDITION

THE present treatise is the final volume of the author's work on the diagnosis and treatment of the diseases of the gastro-intestinal tract. In this book I have closely followed along the lines laid down in my earlier work on Diseases of the Stomach. It has been my aim throughout to meet the requirements of the general practitioner.

Without neglecting those diseases which are generally met with in hospital practice, I have given special prominence to the affections which the private practitioner is called upon to treat—e. g., intestinal catarrhs and ulcers, duodenal ulcer, chronic constipation, rectal diseases, intestinal neuroses, etc. I have devoted no space to the discussion of intestinal parasites, and would refer the reader to standard text-books of medicine or the numerous monographs on the subject.

In many of the chapters I have drawn upon my own experience as gained both in a large polyclinic and hospital practice. I hope that I have been able to add some new and perhaps valuable facts to the pathology and treatment of intestinal diseases.

While physical methods have been so thoroughly studied that only technical differences remain, examination of the fæces has heretofore been very much neglected. Whereas putrid sputa, badly smelling lochia, offensive secretions of uterine cancer in themselves no longer offer serious objection to examination, most physicians cannot accustom themselves to the analysis of the intestinal dejecta. This may in part be due to the circumstance that deductions can be drawn only after repeated careful examinations. This latter consideration should not, however, influence the conscientious physician, for urine and sputum, too, are very often examined with negative results.

Since many intestinal diseases may require surgical interference at any moment, I have felt it necessary in a number of chapters to define my position in this respect. As an internal practitioner I have naturally little sympathy with extreme radical measures, and, with increasing experience, believe with conservative surgeons that we have almost reached the limits of possibility in intestinal surgery.

As in diseases of the stomach, abdominal surgery has also made considerable advance within the last decade in diseases of the intestines. If the medical practitioner wishes to keep abreast of progress he must follow these advances with the greatest conscientiousness, and consider carefully the changes made from time to time in surgical technic, noting the results obtained therefrom. Should he have the good fortune to be associated with a skilful abdominal surgeon, he should use every opportunity to witness operations upon the intestine. This sharpens the judgment, demonstrates the knowledge or ignorance of surgery, and indicates to us, even better than cumbersome and frequently coloured statistics, the manner in which we must proceed in serious cases. As I have already stated in another place, I must again emphasize that the indications for operative procedures in diseases of the stomach and the intestines, as well as of the liver and gall bladder, is a matter which rests mainly with the medical practitioner. He should bear the responsibility for the operative interference, while the surgeon should be responsible for the technic. The placing of the responsibility in one's hands implies the greatest confidence on the part of the patient and his family, and the medical practitioner can only accept such responsibility when he has recognised the disease in time, and is in a position to judge whether or not a surgical procedure is indicated, and with what prospects of success.

In discussing these difficult and very important questions, I have agreed in most respects with the views of early writers.

In stating my views I have endeavoured to include theirs. In this connection I feel called upon to express my admiration for the epoch-making treatise on intestinal diseases of Professor Nothnagel, of Vienna, and my appreciation of his classical studies on the physiology and pathology of the intestines.

Finally, I take great pleasure in acknowledging my thanks to my publisher, Mr. George Thieme, of Leipsic, for the careful preparation of the work. I desire also to thank Miss Paula Günther, of Berlin, and my former assistant, Dr. Reitzenstein, of Nürnberg, for the excellent execution of the drawings.

THE AUTHOR.

BERLIN, *July, 1899.*

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INTRODUCTORY

CHAPTER I

*PRELIMINARY ANATOMICAL AND HISTOLOGICAL REMARKS**

THE intestinal canal is that portion of the alimentary tract which is situated below the pylorus. The small intestine has for its main function the digestion of unassimilated food; the large intestine serves for the reception and propulsion of undissolved and waste food products until their expulsion from the body. Occasionally the large bowel, especially the rectum, is called upon to digest and absorb nourishment, but this vicarious process of nutrition is not adequate to support life a long time.

SMALL INTESTINE

The small intestine, a small, thin-walled tube about 7 metres long, extends from the pylorus to the right iliac fossa, where, after becoming extremely convoluted, it finally opens into the large intestine. It is divisible into three portions, which vary as regards length and calibre—viz., duodenum, jejunum, and ileum.

1. DUODENUM

The duodenum, about 30 centimetres long and 4 to 6 centimetres wide, is the widest and at the same time least movable portion of the small intestine. It describes a horse-shoe curve, the convexity of which is directed toward the right and downward, its concavity embracing the head of the pancreas. Owing to its peculiar form the duodenum is divided into three segments: superior horizontal portion (*pars horizontalis superior*), a descending portion (*pars descendens*), and an inferior ascending portion (*pars horizontalis inferior*) (*seu oblique ascendens, seu transversa*).

The *pars horizontalis superior*, the shortest division (5 centimetres long), commences at the pylorus at the level of the first lumbar vertebra. From here it passes slightly upward, backward, and to the right, thus gaining the right side of the vertebral column. Ascending to the neck of the gall bladder, it then bends abruptly downward, and is continued as the *pars descendens*. The superior horizontal portion is invested by peritoneum, both anteriorly and posteriorly, and behind is in relation with the hepatic duct and the

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* In writing the present chapter use has been made of the more popular text-books of anatomy, especially A. Rauber's Lehrbuch der Anatomie, Leipzig, 1892.

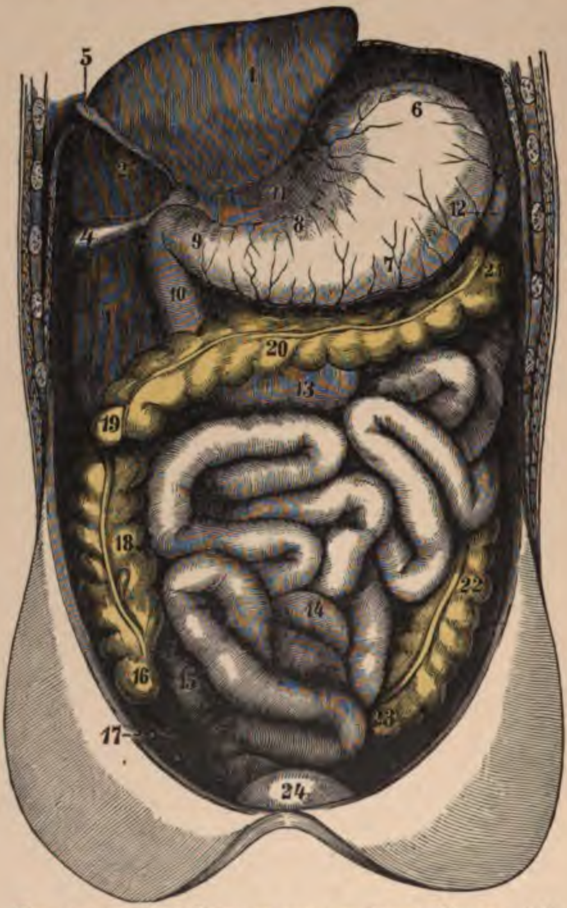

FIG. 1.—ANTERIOR VIEW OF THE ABDOMINAL VISCERA ($^1/_5$).

(The liver is turned upward, thereby drawing the stomach and duodenum slightly upward and to the right.) *1*, left lobe of liver; *2*, lobus quadratus; *3*, right lobe of liver; *4*, gall bladder; *5*, round ligament of liver; *6*, fundus of stomach; *7*, greater curvature; *8*, lesser curvature; *9*, horizontal portion of duodenum; *10*, descending portion of duodenum; *11*, lesser omentum; *12*, spleen; *13*, jejunum; *14*, ileum; *15*, ascending loop of ileum; *16*, cæcum; *17*, vermiform appendix; *18*, ascending colon; *19*, hepatic flexure; *20*, transverse colon; *21*, splenic flexure; *22*, descending colon; *23*, sigmoid flexure; *24*, bladder. (Rauber.)

blood-vessels passing to the liver (the portal vein and the hepatic artery). This part frequently is found stained with bile.

The *pars descendens duodeni*, which begins at the neck of the gall bladder, is twice as long as the first portion. It passes almost vertically downward in front of the right kidney and to the right of the vertebral column, about as far as the third or fourth lumbar vertebra. The transverse colon crosses at right angles in front of it. The common bile duct opens into the descending portion just before the latter merges into the *pars horizontalis inferior*. The duct descends behind the left border of the descending portion, and then, together with the pancreatic duct, which accompanies it for a short distance, penetrates the wall of the gut. Thus a kind of longitudinal swelling is formed, at whose lower end the common opening of the ducts is situated. This opening is frequently hollowed out—the *diverticulum Vateri.*

The lower transverse portion—the *pars horizontalis inferior*, or, more correctly, the *pars ascendens*—equals or even exceeds in length the portion just described. It ascends obliquely from right to left, reaching the left side of the second lumbar vertebra, where, making a sharp bend—the *flexura duodeno jejunalis*—it merges into the jejunum. It passes behind the origin of the transverse mesocolon and the mesentery, while the abdominal aorta and the vena cava lie in front of this division. A short, fibrous, muscular strand, derived from the left crus of the diaphragm (the suspensory muscle of the duodenum), retains this portion of the duodenum in place. Thus, in contrast to the stomach, and especially to the large intestines, the duodenum is usually fixed; nevertheless, as a result of marked distention and traction, it may descend to a greater or lesser degree.

2. Jejunum and Ileum

The jejunum and ileum, which together have received the name of *intestinum mesentericum*, merge into each other without any sharply defined line of separation. Formerly the term jejunum was applied to those portions of the small intestines which lie in the umbilical region and in the left iliac fossa, while the ileum included the portions in the right half of the abdomen, in the right iliac fossa, and in the pelvis. There are no marked differences in the structure of these two divisions. According to Hyrtl, three fifths of the small intestine below the duodenum constitute the jejunum and the remaining two fifths the ileum. The great mobility of the small intestines does not permit of any constant position of the coils, but in general the upper coils lie more transversely and the lower more vertically. This mobility, however, is of the greatest practical importance; it allows of the adaptation of the intestines to the most diverse conditions of the abdominal cavity, and also of their gliding aside when the cavity is filled with serous or other effusions.

The mesentery, which binds the ileo-jejunum to the spinal column, is of great importance. Fan-shaped, it spreads from its origin (*radix mesenterii*) and lies in many folds. Its edges are attached to the small intestines by means of a small slip, the mesenteric border.

Blood-vessels, lymphatics, and nerves run between the two layers of the mesentery. They enter the wall of the intestines at the mesenteric border and terminate at the opposite free side.

The *arteries* supplying the duodenum are derived partly from the cœliac axis and partly from the superior mesenteric artery, as follows: The cœliac axis gives off the hepatic artery, which supplies the liver. The gastro-duodenal branch of the hepatic artery passes behind the stomach at the junction of the pylorus and the *horizontal* portion of the duodenum, and in turn gives off the pancreatico-duodenalis superior, which supplies the duodenum and the pancreas. In addition to the latter vessel the duodenum is also supplied by the pancreatico-duodenalis inferior, a branch of the superior mesenteric artery. The latter vessel passes beneath the pancreas, while its branch (the pancreatico-duodenalis inferior) winds upward and to the right, passing between the lower half of the duodenum and the head of the pancreas, and ultimately anastomoses with the pancreatico-duodenalis superior.

The jejunum and ileum are supplied by branches of the superior mesenteric (*arteriæ intestinales*). They pass between the layers of the mesentery, dividing forklike, and finally form a rich capillary network throughout the entire intestinal wall. The lower end of the ileum alone receives its blood supply from branches of the ileocolic (colica-dextra), which latter, in part, also supply the cæcum and the vermiform process, and, in conjunction with the superior mesenteric, the lower end of the ileum.

Thus, we see, a dense network of the most delicate blood-vessels extends throughout the entire mesentery of the small intestines, piercing the muscular layers of the gut and penetrating to the submucosa, where a second network is formed which supplies the mucous membrane, the folds, villi, and glands of the mucosa.

The *veins* which carry the blood from the intestines into the portal vein have a course differing somewhat from their corresponding arteries; viz., the branches corresponding to the gastro-duodenal artery (i. e., the gastro-epiploica and the pancreatico-duodenal) empty into the superior mesenteric vein.

The *lymphatics* may be divided into two sets, a superficial and a deep. The superficial originate in the muscularis (subserous lymphatics), while the deeper originate in the mucous membrane, the villi, and the solitary follicles (submucous lymphatics). Both sets unite at the mesenteric border of the small intestine and then pass between the layers of the mesentery. Owing to their physiological significance, they are called lacteals.

The *nerves* of the small intestines are derived chiefly from the superior mesenteric plexus of the sympathetic. The hepatic plexus, an offshoot of the cœliac plexus, gives branches to the duodenum. Furthermore, the small intestine is supplied by the abdominal portion of the vagus; viz., the anterior and posterior gastric plexuses. The nerves, which are for the most part non-medullated, accompany the branches of the superior mesenteric artery to the intestinal wall, and there form a subserous meshwork; they then pierce the longitudinal muscular layer, forming between the latter and the circular layer a network consisting of numerous multipolar cells—the mesenteric plexus of Auerbach.

From the latter delicate nerve branches supply the muscularis; others penetrate the circular muscular layer to the submucosa, where they form the submucous or Meissner nerve plexus—a very fine network of nerves containing small ganglion-cell groups. Bundles of nerve fibres pass from this plexus to the muscularis mucosæ and to the muscularis of the villi, and are then lost in the mucous membrane.

Histology of the Small Intestines

The wall of the small intestines is composed of four coats: tunica serosa, muscularis, submucosa, and mucosa (Fig. 2, page 6).

The *serous coat* (peritoneum), as has been already mentioned (page 2), does not uniformly invest the small intestines. Practically speaking (and this is especially of surgical interest), the descending portion of the duodenum is covered only upon its anterior surface by the serous layer, while the superior and inferior horizontal portions are inclosed by both folds of the mesocolon. The serous layer is most adherent at the free border of the intestines, and but loosely adherent at the mesenteric border.

The *muscularis* of the intestines consists of two layers of unstriped muscle fibres: a thick inner circular, and a thin external longitudinal layer. Toward the ileum the layers gradually become thinner.

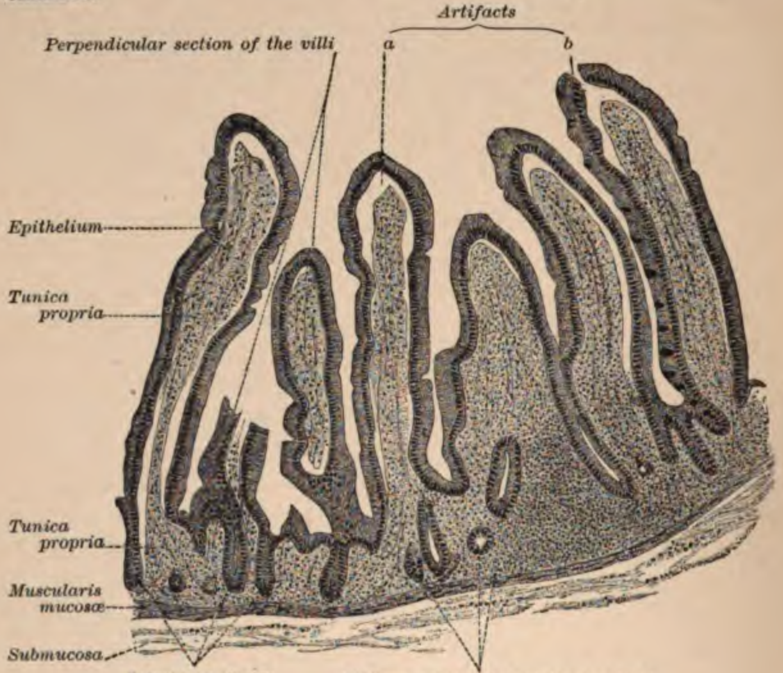

FIG. 2.—PERPENDICULAR SECTION OF ADULT HUMAN JEJUNAL MUCOUS MEMBRANE (× 80).

During fixation the tunica propria of the villi retracted and became separated from the epithelium, thus causing a space at *a* and a tear at *b*. The dark spots in the villi, at the right-hand side of the figure, are goblet cells. (After Stöhr.)

The *submucosa* is composed of loose retiform tissue, and supports the above-described numerous blood and nerve plexuses. The *muscularis mucosæ* consists of smooth muscle fibres, an inner circular and an outer longitudinal layer. From it fibres pass perpendicularly and inwardly, almost reaching the apices of the intestinal villi. By their contraction they may cause shortening of the villi.

The *epithelium* of the mucosa (Fig. 3) consists of a single layer of cylindrical cells. We may distinguish two forms: cylindrical epithelium with a basement membrane, and the goblet cells. The significance of this basement membrane is still a matter of controversy. According to the latest investigations, we must here recognise a very delicate skeletal framework with free interspaces; through these latter, very fine protoplasmic prolongations of the epithelial cells can be thrust and again withdrawn. Thus, the chief seat of absorption is evidently in the basement membrane of the epithelial cells. The goblet cells have an oval, not infrequently a gobletlike, form, the upper (free) part being more or less filled with mucus, which results from a protoplasmic metamorphosis, while the nucleus lies at the base of the cell. They have no basement membrane, but have a sharply defined opening at their free border, through which the mucus is poured into the intestines. Leucocytes are present in varying numbers between the epithelial cells.

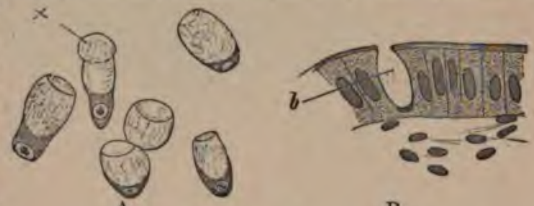

FIG. 3.—INTESTINAL EPITHELIUM (× 560).

A, goblet cells of rabbit (at × protrusion of mucus); B, portion of a section of human small intestine; b, a goblet cell between cylindrical cells. (Stöhr.)

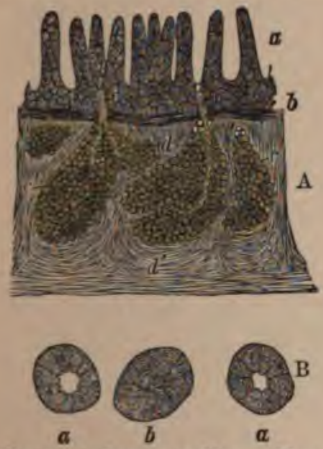

FIG. 4.—A. SECTION OF MUCOUS MEMBRANE OF HUMAN DUODENUM (× 10).

a, villi; b, basement substance of the mucous membrane; c, Brunner's glands; d', deepest layers of the submucous tissue.

B. TRANSVERSE SECTION OF ISOLATED GLANDS.

a a, with lumen; b, without lumen. (Rauber.)

The *tunica propria* consists mainly of reticular connective tissue, with here and there numerous leucocytes. Owing to the size and number of the glands of the large intestines, the tunica propria is insignificant forming little more than the intervening substance between the glands and a narrow strip of basement substance. In the small intestines the tunica propria forms numerous cylindrical projections from the inner surface of the gut, the projections being 0.5 to 0.7 millimetre in height and 0.1 to 0.2 millimetre in width; these are the so-called intestinal villi (Fig. 4). In the duodenum they are leaflike in shape. To a certain extent their functions are like those of the roots of trees, inasmuch as they dip directly into the nutritive material in the intestinal canal, and absorb all of it thereof that can be taken up (A. Rauber). The total number of villi is estimated as over ten million. Each villus contains a central chyle space, or villous sinus, which is a club-shaped expansion of the lacteals of the intestinal mucous membrane, and is lined with endothelium. The larger villi contain several of these spaces. The blood-vessels of the villi spread out in the reticular tissue between the external and internal endothelium. This rich capillary network may cause erection of the villi, while the previously mentioned offshoots from the muscularis mucosæ cause their rhythmical contractions. *Thus the villi act as simple and yet complete suction pumps.* The villi are most numerous in the duodenum, gradually diminishing in number in the ileum. Each villus receives its blood supply from one or more arterial branches which, dividing, form a meshwork near the epithelium, from which meshwork the corresponding vein arises.

A similar arrangement, intended for the greatest possible absorption and a uniform distribution of nutritive material, is found in the so-called folds of Kerckring (*valvulæ conniventes Kerckringii*). These occupy one half to two thirds of the transverse circumference of the mucous membrane, and are found close together in the upper third of the small intestine. They are absent in the upper third of the transverse portion of the duodenum. They number about eight hundred, and the distances between the individual folds, according to Sappey, average 15 millimetres. In the upper third of the small intestines their height and the intervals between them are uniform; in the middle they diminish both in height and breadth, and finally disappear in the lowermost coils of the ileum.

Glands form another constituent of the mucous membrane of the small intestines. The liver and the pancreas should be reckoned among these, since their secretions form an important—indeed, an indispensable—part of the intestinal juices. Since these are independent abdominal organs, their structure can not be entered into here. We have already discussed the openings of the ducts of these glands (page 3). The glands proper of the intestines are of two varieties: secretory and agminated. The first variety includes Brunner's and Lieberkühn's glands; the second variety includes the so-called solitary blind follicles and Peyer's agminated glands (Peyer's patches). Brunner's glands (Fig. 4), which are found almost exclusively in the upper part of the duodenum, are spread over an area of from 8 to 10 centimetres from the pylorus. They are conglomerate tubular glands whose bodies lie within the submucosa. Their terminal portions are lined with cylindrical cells having a lightly coloured granular protoplasm and an oval nucleus which lies near the periphery. The cells are best found by dissecting away the muscularis from without.

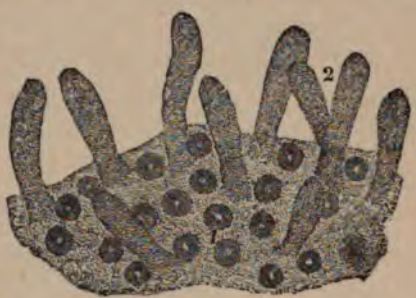

FIG. 5.—SURFACE OF MUCOUS MEMBRANE OF THE SMALL INTESTINE.

1, openings of Lieberkühn's glands; *2*, villi. (Rauber.)

In structure, Lieberkühn's glands (Figs. 5 and 6) resemble closely the tubular glands of the stomach; they are evidently the true secreting glands of the small intestines. They are present in enormous numbers throughout the mucous membrane of both small and large intestines. They are club-shaped and rounded off both above and below. Like the glands of the stomach, they are also seldom branched. They measure from 0.3 to 0.4 millimetre in length, their total number being estimated by Sappey at forty to fifty million. According to Drasch, the glands are surrounded by a fine network of capillaries and nerves. They generally terminate in circular openings between the villi, and when viewed with a lens give the mucous membrane a honeycomb appearance. As is well known, the solitary lymph nodules (solitary follicles) are also met with in the œsophagus and stomach. They are quite uniformly distributed throughout the surface of the small intestine. They have an elongated oval shape and are as large as millet seeds, but under pathological conditions may reach the size of a pea, or even larger. They extend deeply below the submucosa. As regards their finer structure, this consists of adenoid tissue and usually contains a germinal centre. The leucocytes so frequently present in the follicles may pass into the lymphatic vessels or, by piercing the epithelium, may enter into the lumen of the intestines.

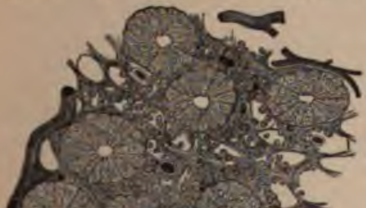

FIG. 6.—CROSS SECTION OF INTESTINAL MUCOUS MEMBRANE (× 150).

Showing Lieberkühn's glands with their epithelial cell-lining embedded in the adenoid tissue of the mucous membrane, from which the cells are partially absent. (Rauber.)

Peyer's patches occur in the ileum as elongated plaques, from 2 to 10 centimetres in length and from 1 to 3 centimetres in breadth, their long axis corresponding to that of the gut. They are never situated at the mesenteric border. Occasionally they are met with in the jejunum, or even higher up, in the duodenum. Usually twenty to thirty such plaques are present. They are made up of groups of solitary nodules spread out over a flat surface (Fig. 7); occasionally they become flattened from pressure. The mucous membrane covering the glands is, as a rule, thrown into folds, but it has no villi. Villi are, however, frequently present as flat folds upon the intervening elevations (Henle).

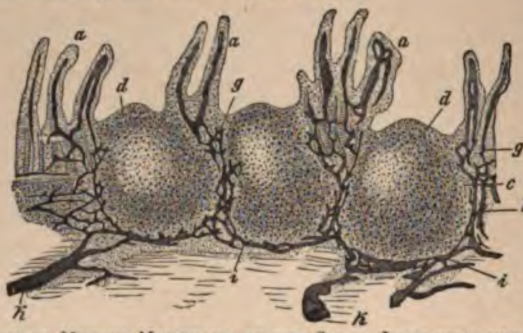

FIG. 7.—SECTION OF MUCOUS MEMBRANE OF THE SMALL INTESTINE THROUGH A PEYER'S PATCH, THE CHYLE VESSELS BEING INJECTED.

a, villi; *c*, follicles; *d*, projections of the latter toward the surface; *g*, *h*, *i*, lymphatic network around the follicles; *k*, efferent blood-vessels. (Frey.)

The branches of the chyle vessels form numerous meshes about the glands, and communicate with them by means of very delicate projections. These projections permit the passage of newly formed lymph cells into the lymph channels, for Peyer's patches are breeding places for lymph cells (A. Rauber).

LARGE INTESTINE

The large intestine (Fig. 8, page 11), the lower division of the intestinal canal, commences in the right iliac fossa, and thence ascends along the right posterior wall of the abdomen to the right hypochondrium. Here it comes in contact with the under surface of the liver, and, bending to the left, passes transversely and slightly upward to the spleen. There, in the left hypochondrium, it bends downward and descends along the left side of the abdomen to the left iliac fossa, and thence onward into the pelvis. The large intestine measures about 1.5 metres in length (according to Sappey, exactly 1.68 metres), and varies in diameter from 5 to 8 centimetres. Its diameter gradually diminishes from the cæcum downward. The dimensions just given may be greatly altered by pathological conditions.

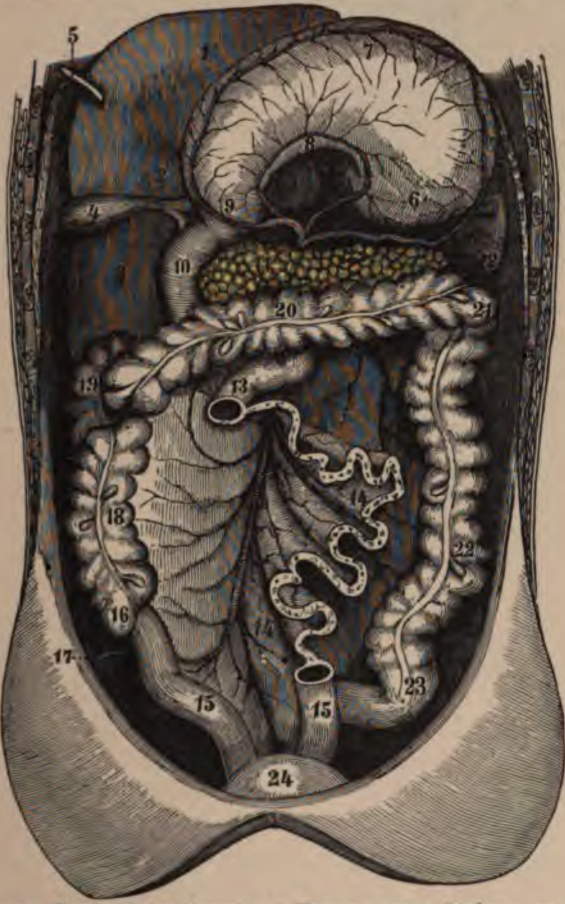

FIG. 8.—ANTERIOR VIEW OF THE ABDOMINAL VISCERA AFTER REMOVAL OF THE JEJUNUM AND ILEUM (× $^1/_8$).

(Liver and stomach are turned up, the jejunum and ileum, excepting their two ends, removed, the mesentery is retained). *1*, left lobe of liver; *2*, lobus quadratus; *3*, right lobe of liver; *4*, gall bladder; *5*, round ligament of liver; *6*, fundus of stomach; *7*, greater curvature; *8*, lesser curvature; *9*, pylorus; *10*, duodenum: *11*, pancreas; *12*, spleen; *13*, jejunum; *14*, mesentery; *15*, ileum; *16*, cæcum; *17*, vermiform appendix; *18*, ascending colon; *19*, hepatic flexure; *20*, transverse colon; *21*, splenic flexure; *22*, descending colon; *23*, sigmoid flexure; *24*, bladder. (Rauber.)

The large intestine differs from the small not only in calibre but also by having three narrow longitudinal bands of unstriped muscle fibres (*ligamenta* or *tænia coli*). These begin in the cæcum at a point corresponding to the insertion of the vermiform appendix. Through them the intestine is thrown into numerous sacculi (*haustra coli*), separated from one another by deep folds (*sulci transversi*). These folds and pouches do not exist in the rectum, hence the latter is readily distinguished from the other portions of the large intestine.

As regards its course, the large intestine is divisible into

1. A blind, pouchlike commencement, the *cæcum*, with the vermiform appendix.
2. An ascending portion, *colon ascendens*.
3. A transverse portion, *colon transversum*.
4. A descending portion, *colon descendens*.
5. The portion within the left iliac fossa, *flexura sigmoidea* (*S. romanum*).
6. The rectum.

Each of these subdivisions requires separate consideration.

1. Cæcum and Vermiform Appendix

The blind gut is that portion of the large intestine lying immediately below the termination of the ileum. Its length is subject to wide fluctuations, variously estimated from 4 to 12 centimetres. According to Henle, the average length is 5.5 centimetres. Its width almost equals its length. The cæcum lies in the right iliac fossa above the middle of Poupart's ligament, and is in contact anteriorly with the abdominal wall. When immoderately long, it may extend into the small pelvis. In the majority of cases the cæcum is completely invested by peritoneum (the mesocæcum). This would account for its great freedom of motion and the frequency with which it forms one of the contents of femoral and inguinal hernia. In rare cases the peritoneum is absent from its posterior surface. Attached to the anterior surface of the lower middle division of the cæcum we find the vermiform appendix, an organ of extreme practical importance, but of whose physiological functions we at present know nothing. It varies in length from 2 to 20 centimetres; its width is about ½ to 1 centimetre. Usually it is spiral-shaped, and is directed from the right iliac fossa toward the border of the small pelvis, or it may even dip into the small pelvis. It has a small mesentery (*mesenteriolum*) and, like the cæcum, it is freely movable. The appendix is hollow up to its apex, and communicates with the cæcum through a small opening, the *ostium processus vermiformis.* This opening is sometimes guarded by a small crescentic fold, the *valvula processus vermiformis.*

The terminal opening of the small intestine is found at the junction of the cæcum and ascending colon. This opening, which leads from the ileum into the large intestine, is guarded by a valve formed of two crescentic folds, the *valvula coli* or *Bauhini* (Fig. 9). The two folds, superior and inferior, are united at their ends, but in the centre form an aperture at right angles to the long axis of the colon. Normally this valve permits the passage of contents from the small intestines into the large, but not conversely. It will not even allow the passage of gases insufflated into the large intestine. Only the combination of extreme pressure with a relative insufficiency of the valve, such as occurs in paresis of the intestinal wall, can effect a passage of intestinal contents into the ileum.

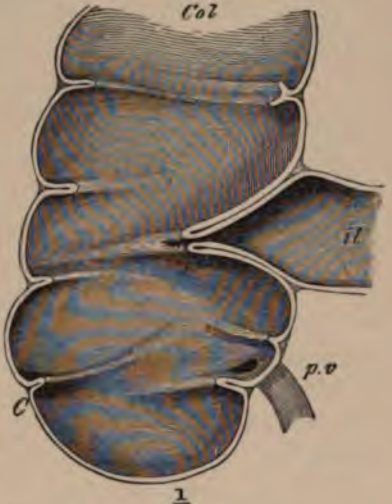

FIG. 9.—OPENING OF THE ILEUM INTO THE LARGE INTESTINE.

(Perpendicular section through the cæcum and ileo-cæcal valve.) *p. v.*, vermiform appendix, whose opening into the cæcum is visible. (Gegenbaur.)

2. ASCENDING COLON

The ascending colon passes almost vertically upward from the right iliac fossa to the under surface of the liver, upon which latter surface it produces the *impressio colica.* In the region of the gall bladder the ascending segment of the colon leaves the posterior abdominal wall and passes sharply forward and to the left, thus becoming more superficial; then, forming the *hepatic flexure*, the gut continues on as the *transverse colon.* The hepatic flexure of the colon is connected with the liver by a short, taut band, the *ligamentum hepatico-colicum.* In front, the ascending colon is in relation with coils of the small intestines; behind, with the lateral border of the quadratus lumborum and with the transversalis abdominis, as well as with the lower anterior surface of the right kidney. Therefore it is possible to have nephro-colic abscesses without peritonitis.

The remainder of the ascending colon is completely surrounded by peritoneum.

3. Transverse Colon

The transverse colon passes from the right hypochondrium upward and to the left behind the anterior abdominal wall to the left hypochondrium; then, bending acutely, it continues as the *descending colon*. The angle thus formed is known as the *splenic flexure*. At this flexure the colon leaves its superficial position and passes sharply downward and backward. The flexure is connected with the diaphragm by the *ligamentum phrenico-colicum*. The transverse colon is provided with a very long mesentery, the *mesocolon transversum*, and is therefore freely movable. This point will again be referred to.

4. Descending Colon

The descending colon passes from the splenic flexure vertically downward through the left hypochondriac and lumbar regions to the left iliac fossa, where it forms an **S**-shaped fold, the *sigmoid flexure*. The splenic flexure is in contact above with the spleen. Anteriorly, the descending colon is covered for the most part with coils of small intestine. The descending colon, like the ascending, has no mesentery of its own, and it therefore is not freely movable. It is invested only laterally and anteriorly by peritoneum. Its posterior surface is in relation with the costal portion of the diaphragm, with the left kidney, the transversalis abdominis and quadratus lumborum muscles, as well as with the iliac fascia, to all of which structures it is connected by loose connective tissue.

5. Sigmoid Flexure (S. Romanum) (Fig. 10, page 17)

This is formed by a double loop of the colon, and has the appearance of an inverted S. We may distinguish an upper (colon) segment whose convexity is directed toward Poupart's ligament, and a lower (rectal) segment which projects more or less into the pelvis. As a rule, the transition to rectum occurs at the sacro-iliac synchondrosis.

The sigmoid flexure is entirely covered by peritoneum, which forms a rather long mesentery, the *mesocolon flexuræ sigmoideæ*. On this account the flexure is freely movable. In the newly born the sigmoid flexure has a very long mesentery, and it may therefore lie on the right side near the cæcum, especially if defecation has not yet occurred and the flexure is still filled with meconium.

Blood-vessels, Lymphatics, and Nerve Supply of the Large Intestine

The blood supply of the large intestine is derived from the three colic vessels, the left colic artery, a branch of the inferior mesenteric artery, and the middle and superior colic arteries, branches of the superior mesenteric artery.

The *veins*, which have a course parallel to that of the arteries, empty into the superior and inferior mesenteric veins.

According to Sappey, the *lymphatics* of the intestinal wall are numerous and form two sets—a deeper, beneath the glands of Lieberkühn, and a more superficial, which forms a network ramifying in all directions in the submucosa.

The *nerves* which supply the cæcum, ascending colon, and the right half of the transverse colon are derived from the superior mesenteric plexus, which is given off by the cœliac plexus. The left half of the transverse colon, the descending colon, and the sigmoid flexure receive their nerve supply from the inferior mesenteric plexus, which in its turn is derived from the plexus of the abdominal aorta.

Histology of the Large Intestine

Like the wall of the stomach and small intestines, that of the large intestine is composed of four layers: serous, mucous, submucous, and muscular.

The *serous* coat has already been considered in speaking of the separate segments of the intestine.

The *muscular* coat consists of an external longitudinal and an internal circular layer. The longitudinal layer is not found throughout the entire gut, but appears as three broad longitudinal bands, *tænia* (*ligamenti*) *coli*, which are each 10 millimetres in width and 2 to 3 millimetres in thickness, and are all visible through the serosa. Beginning at the attachment of the appendix, they continue as separate bands as far as the rectum, where they unite to form a continuous muscular layer.

One of these bands is seen at the attachment of the gastro-colic omentum of the transverse colon; at its mesenteric border is a second, while the third band is free. Hence they are known as the *tænia omentalis*, *mesenterica* and the *libera*. Between them there is a triple row of alternately protruding and receding areas. The protruding parts are termed the *haustra coli;* the depressions, which are parallel to each other and perpendicular to the plane of the wall of the gut, are known as the pockets. Wherever haustra and tænia cross, we find projections of the serous coat, rich in fat, called *appendices epiploicæ*.

The *circular* coat extends over the entire colon; it is strongest between the haustra, which it surrounds by folds of considerable thickness (*plicæ sigmoideæ*).

The *submucosa* is entirely like that of the small intestines.

The *mucous* membrane differs from that of the small intestines, above all, through the absence of Kerkring's folds and of villi. It is thicker than that of the small intestine. The *muscularis mucosa*, a thin layer of crossed muscular fibres, lies beneath the mucosa. The glands of Lieberkühn lend a sievelike appearance to the mucous membrane of the large intestine. These glands resemble the glands of the small intestine, but are longer and more frequently branched than the latter. The mucous membrane between Lieberkühn's glands contains cylindrical epithelium, goblet cells, and numerous solitary lymphatic nodules.

6. The Rectum

We include in the term rectum the lower segment of the large intestine from the sigmoid flexure to the anus. The rectum commences at the sacro-iliac synchondrosis, and therefore lies entirely within the pelvis. It varies in length from 18 to 22 centimetres (according to other authorities, 25 to 33 centimetres). Its lumen is narrower than that of the sigmoid flexure; immediately above its termination there is a normal dilatation, the *ampulla recti*, which even normally varies largely in size. Contrary to its name, the rectum is not straight, but presents curves, three of which can be distinguished. Beginning at the sacro-iliac articulation, it at first passes slantingly downward and to the right, then runs forward in front of the lower portion of the sacrum and coccyx, being (in men) behind the bladder, the seminal vesicles, and the prostate, or (in women) behind the cervix of the uterus and the vagina. Behind the last-named organs (or the prostate) the rectum again curves downward and backward and terminates at the anus.

For practical purposes we distinguish between the rectum proper and the anal portion—i. e., the part surrounded by the sphincter ani.

The upper portion only of the rectum is covered by peritoneum, the *mesorectum*, a direct continuation of the *mesocolon sigmoidea*.

Like the sigmoid, the upper portion of the rectum has also some range of motion. Farther down, the peritoneum recedes from the rectum and is reflected on to the bladder in the male, or on to the vaginal vault and uterus in the female. Above the point of reflection two crescentic folds, inclosing smooth muscle fibres, pass from the sides of the rectum to the bladder or to the uterus (the folds of

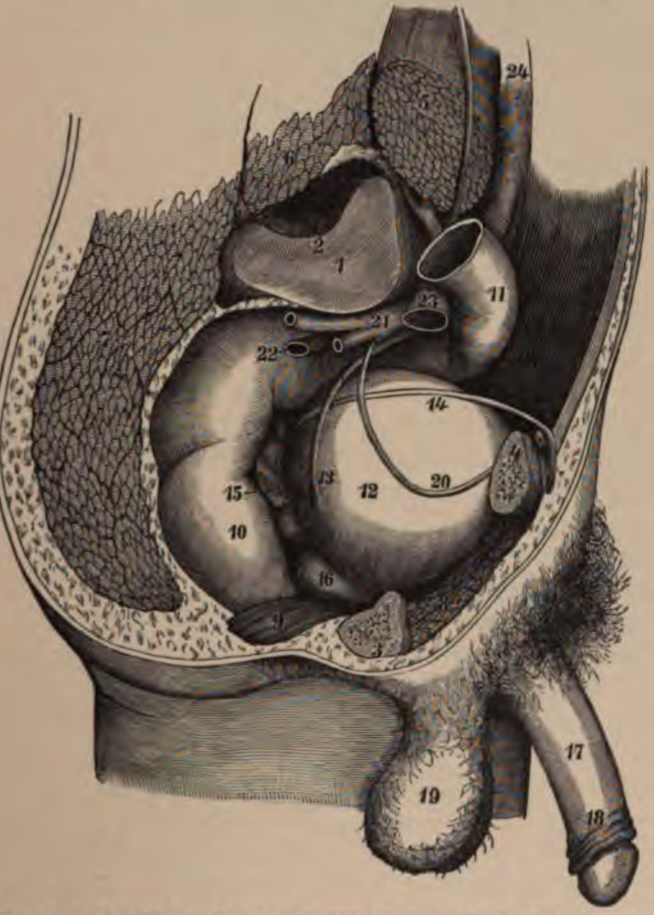

FIG. 10.—MALE PELVIC ORGANS, VIEWED FROM THE RIGHT SIDE (× 1/3).

(The right ilium and a portion of the ischium and the pubic bone, together with their soft parts, have been removed.) *1*, auricular surface of the sacrum; *2*, tuberosity of the sacrum; *3*, ischium; *4*, pubic bone; *5*, psoas muscle; *6*, erector spinæ; *7*, glutei muscles; *8*, obturator muscles; *9*, external sphincter of anus; *10*, rectum; *11*, sigmoid flexure; *12*, bladder; *13*, ureter; *14*, vas deferens; *15*, seminal vesicles; *16*, prostate; *17*, penis; *18*, prepuce; *19*, scrotum; *20*, lateral vesical ligament; *21*, hypogastric artery; *22*, hypogastric vein; *23*, external iliac artery; *24*, abdominal aorta. (Rauber.)

Douglas). The space beneath these folds is known as the *recto-vaginal*,and the one above as the *recto-vesical*, *pouch*.

The relative position of these peritoneal folds is not constant, however, but varies according to age and sex, as well as to special conditions of the neighboring organs. The above subdivision is therefore of an anatomical rather than a practical value (von Esmarch).

The *muscular coat* of the rectum consists of an inner circular and an outer longitudinal layer. The circular layer, which is directly continuous with that of the colon, constantly increases in thickness toward the lower end of the gut, finally forming a ring 1 or 2 centimetres in length, the *internal sphincter of the anus*. The external, the longitudinal, layer is continuous with the three longitudinal bands of the colon. In the rectum it forms a uniform layer, interrupted here and there by small intervals. The fibres become thinner in the lower part of the rectum, and finally merge with those of the external sphincter.

Contrasted with the feeble muscular coat of the upper portion of the rectum, the anal portion contains two powerful bands of circular fibres which secure closure of the anus. They are the external and internal sphincters (*sphincter ani externus et internus*).

The *external* sphincter, whose action is voluntary, arises by a tendinous origin from the apex of the coccyx, and separates into two divisions which surround the anal orifice. In front of the anus it is connected, in the male, with the musculus bulbo-cavernosus, or transversus perinei, and in the female with the constrictor cunni.

The *internal* sphincter of the anus, a ring or girdle of smooth muscle fibres, is the direct prolongation of the circular layer of the rectum; these fibres gradually increase in strength as they approach the anus. Connected with the internal sphincter are two muscular fasciculi, 4 millimetres in width, which spring from the anterior surface of the coccyx and surround the lower end of the rectum (the *musculi recti-coccygei*).

A short distance below the middle of the rectum (about 8 centimetres above the anal orifice) we sometimes find a prominent collection of circular fibres, to which the name "*sphincter ani tertius*" has been given (Hyrtl). This "sphincter," however, is nothing more than the transverse rectal fold increased by some circular fibres.

The mucous membrane of the rectum is thicker, redder, and more succulent than that of the colon. It presents many folds, varying in size and directions, and disappearing when the organ is strongly distended. They are most distinct when the rectum is empty. One deep, transverse fold alone does not disappear even with the greatest possible distention of the rectum. This fold—the *plica transversalis recti*—is about 6 to 8 centimetres above the anus, and can be distinctly seen through a speculum. It is a sickle-shaped reduplication of the mucous membrane, and does not include the entire circumference of the rectum. Near the anal opening the folds are usually longitudinal, and have received the name of rectal or *columns of Morgagni*. The pockets between the folds are known as *sinuses of Morgagni;* they are favourite lodging places for intestinal parasites and for swallowed pointed foreign bodies. The mucous membrane covering these folds and pockets differs from that of the rest of the rectum by the absence of glands and by the presence of moderate-sized papillæ, partly agminated. The epithelium is stratified, and consists of large pavement cells. Thus, the lower portion of the rectum forms a transition from mucous membrane of the intestinal canal to skin (Henle). The mucous membrane of the upper portion of the rectum differs in no respect from that of the remainder of the large intestine.

The rectum receives its blood from five arteries, branches of three arterial trunks. The largest of these five, the superior hæmorrhoidal artery, is given off by the inferior mesenteric; the middle hæmorrhoidal arteries, two in number, come either from the hypogastric or from the common pudendal; the two smallest vessels, the inferior hæmorrhoidal arteries, come from the common pudendal.

The blood is carried away from the rectum for the most part by the portal system through the superior hæmorrhoidal vein, and the remainder by the middle and external hæmorrhoidal veins, which empty into the inferior vena cava.

The *lymphatics* form an extensive network with wide meshes, which sends some branches to the retrorectal glands and others to the left lumbar plexus.

The *nerves* are derived mainly from the sympathetic. They come from the inferior mesenteric plexus, the sacral plexus (inferior and middle hæmorrhoidal nerves), and from the superior hypogastric plexus.

APPENDIX

Displacements of the Various Segments of the Intestines

As a consequence of congenital or acquired anomalies, the various segments of the intestines may undergo alteration in position. These displacements are very important for the diagnostician, and in order to prevent serious error it is necessary that he be acquainted with or at least consider them. The portions most frequently affected are the duodenum and the entire large intestine, exclusive of the rectum; much less frequently, and usually as a consequence of the above anomalies, the remainder of the small intestine.

The superior horizontal portion of the duodenum is so closely connected with the stomach that it would naturally be affected by alterations in position of that organ.* Should the stomach be pushed toward the left side, the superior horizontal portion of the duodenum would be on a line with the pylorus. If the pylorus be dragged to the left of the median line of the body, the first portion of the duodenum will then lie upon the vertebral column. Should the pylorus be within the median line or to the right thereof, the same portion of the duodenum will curve around the vertebral column (Hertz). The gut thus becomes more superficial, and the superior flexure of the duodenum is bent almost at a right angle. Where there is a fish-hook-shaped ptosis of the stomach, the first portion of the duodenum will also be drawn downward, and will assume an almost vertical position. A sharp bend is thus given to the flexure, and it may be so pronounced as to even cause a partial obstruction of the contents. Again, where the stomach *in toto* is sunken, it will drag the first portion of the duodenum, or even the entire duodenum, down with it. Under other conditions the duodenum can preserve its normal position.

The descending portion of the duodenum is less freely movable, because of its close connection with the pancreas. Despite this, both organs may be simultaneously displaced. Braune observed a case in which this portion of the duodenum was dislocated to the left. Displacement of other organs, especially of the stomach, the liver, transverse colon, etc., naturally causes displacement of the duodenum as well. Nevertheless, the tendency of the descending portion to become dislocated is limited.

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* See Hertz. Abnormitäten in der Lage und Form der Bauchorgane, etc., Berlin, 1894, S. 33.

The inferior horizontal portion is subject to slight alterations in position alone, and these are brought about by the same conditions as those of the remainder of the duodenum. We wish here to call attention to the large number of adhesions which occur in the region of the gall bladder and the duodenum, and which may create great difficulties in diagnosis as well as in treatment.

Alterations in position of the colon are most common. Curschmann[1] has recently called attention to their clinical importance. The following statements are taken largely from Curschmann's excellent treatise.

1. The Cæcum and the Ascending Colon

The cæcum may be abnormally long, and thus give rise to volvuli, kinking, and flexures. In such cases the fundus of the cæcum is directed toward the diaphragm, and covers a part of the ascending colon. Thus we may at times have a total occlusion of the gut. It should be remembered that such a dislocated cæcum, together with the appendix, may be the seat of a perityphlitis.

In those very rare cases in which the ascending colon is congenitally entirely absent, the cæcum with its appendix lies very close to the edge of the liver, or behind that organ. Perityphlitis has been observed under such conditions, but the clinical diagnosis is very difficult, if not impossible.

2. The Transverse Colon and the Flexures

It is a well-known fact that the transverse colon [especially in the female] is subject to the greatest variations in position. This is probably due to the habit of lacing, through which one portion of the abdominal contents is forced upward and the other downward. When its mesentery is excessive in length, the transverse colon may be dislocated upward and lie in the epigastrium in front of the liver, or in the left hypochondrium in front of the stomach (Hertz). In other cases, the transverse colon may be entirely concealed by coils of small intestine; we then find it low down in the posterior portion of the abdomen (Hertz).

Absence or shortening of the flexures may give rise to conditions of special interest. For example, the colon may pass from the right side upward and toward the median line; thence transversely for a short distance; then, abruptly bending to the left, con-

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tinue downward as the descending colon. Again, in a very long colon, both flexures may be shortened or absent. The transverse colon in such cases forms a large loop, whose segments lie close to each other and in direct contact with the liver, and may cover the entire anterior surface of that organ. Here we would get apparent diminution in liver dulness in front, but percussion of the lateral and posterior surfaces should save us from mistakes.

Marked increase in length of the large intestine may give rise to the coiling together of the intestines, or even to the formation of a volvulus.

3. The Descending Colon and the Sigmoid Flexure

The sigmoid flexure is the most movable segment of the large intestine, because its length is subject to the greatest variations. Its displacements, as well as those of the descending colon, have been noted by the older anatomists. Recently Schiefferdecker[2] and von Samson[3] have made a close study of the subject.

Schiefferdecker distinguishes the following alterations in position of the descending colon:

I. The end of the descending colon lies to the side of the sigmoid flexure.

(*a*) The flexure projects into the small pelvis, and the small intestines lie in front and above it.

(*b*) The flexure lies upon the posterior abdominal wall, and is directed upward; *a*, it lies within the left half of the body; *β*, it extends to the right iliac fossa. Between these extreme left and right positions the greatest variations occur.

(*c*) Other portions of the intestines separate the sigmoid flexure from the posterior abdominal wall, with the result that a greater or lesser portion of that gut lies directly behind the anterior abdominal wall. These variations can only occur with a free mesosigmoideum, and the higher the lateral point of fixation of the sigmoideum, the greater will be the predisposition to these displacements and their extent upward. The apex of the sigmoid flexure may move from the extreme left side of the body to the extreme right.

II. The termination of the descending colon lies to the median side of the sigmoid flexure. This is extremely rare.

Curschmann, who has given special attention to the clinical importance of the anomalies of the sigmoid flexure, lays particular stress upon the striking differences in its length (60 to 80, or even 100 centimetres). Sometimes there are two coils instead of one. A more frequent anomaly is the formation of an abnormal loop between the lower end of the sigmoid and the beginning of the rectum. In such cases we almost always find the lower end of the colon far down in the pelvis and over to the right side. Under such circumstances, the commencement and termination of the large intestine lie side by side. It is very apparent how gross errors in diagnosis may occur. This has been very aptly illustrated in one of Curschmann's cases. The position of the coils of the sigmoid may also be greatly altered. Normally, they are in the centre of the abdomen, their long axis being parallel with the linea alba. The upper border is in contact with the transverse colon. With increase of length the coils may even reach the vault of the diaphragm, and completely cover the stomach and liver. This abnormality in position may make it difficult to determine the limits of the liver and stomach. It appears that almost invariably the base of the coils is covered by small intestine. If volvulus should occur in these cases, we should then find tympanitic intestinal coils at all points, excepting the one at which the volvulus is situated—i. e., the region of the sigmoid flexure.

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CHAPTER II

PRELIMINARY PHYSIOLOGICAL AND PHYSIOLOGICO-CHEMICAL REMARKS

THE digestive process in the intestines is far more complicated than in the stomach. In the latter the digestive act is but a preparatory one, affecting only the proteid bodies (and gelatinoid substances) and the starches. Absorption, as we now know, takes place but to a limited extent (v. Mering, Brandl, Moritz, Hirsch, and others). The digestive task of the intestines consists principally in converting all food stuffs, brought to it from the stomach, into a form suitable for assimilation; in absorbing the useful portions, and gathering together in a solid form and excreting from the body all waste material. The intestines, however, have another function, heretofore but little appreciated by physiologists and clinicians—i. e., the removal in the form of flatus of noxious gases developed in the intestinal canal. This might be termed intestinal ventilation.

All the functions of the intestines harmonize like the component parts of a score; interference with one deranges all. Strictly speaking, then, these functions should not be considered separately. If, nevertheless, we do so in the following, it is because the entire process of digestion—the influence exerted by a single function upon the whole, and *vice versa*—is not well enough understood either in man or in the lower animals.

In the following we shall first treat separately of intestinal secretion, absorption, and motility, and then of the entire course of intestinal digestion. As by-products of intestinal activity, we have certain fermentation substances, some useful, others harmful. They also will be considered in the section on intestinal digestion in its entirety (page 43).

THE SECRETING FUNCTION OF THE INTESTINES

The secretion furnished by the intestines themselves plays but an insignificant part in the digestive process. The main work is accomplished by the secretions from the two glands emptying into the small intestine, viz., the liver and the pancreas.

1. The Intestinal Juice

This is a mixture of secretions from Brunner's and from Lieberkühn's glands. Grützner[1] ascribes the faculty of secreting pepsin to Brunner's glands; this would give them functions analogous to those of the pyloric glands. They certainly do not secrete any diastatic ferment. Other investigators regard Brunner's simply as mucous or salivary glands.

Our information regarding the secretion of Lieberkühn's glands is more definite, for this has been studied in animals by the aid of the Thiry or Vella fistula, and also in individuals with fæcal fistulæ (Dumont, Turby and Manning). Observations thus made showed the secretion to be pale yellow in colour, highly albuminous and muciform, and containing about 0.5 per cent. of sodium chlorid and a like amount of sodium carbonate. Hoppe-Seyler[2] lays special value upon this mucosity, since mucin forms a protective coating for the intestinal epithelium, and makes possible as well as facilitates the passage of the solid masses within the intestines.

Another very important factor is the presence of the carbonate of soda. This affects the rapid neutralization of the acid stomach chyme. Bunge,[3] in fact, ascribes the importance of the intestinal secretion mainly to its carbonate of soda.*

In dogs, the solids of the intestinal juice equal 12.2 to 24.1 grams per 1,000; in sheep, 46 to 47 grams. According to Thiry, the specific gravity of the secretion varies in dogs from 1010 to 1017.

It is generally conceded that the fermentative action of the intestinal juice is very slight. It has been shown that starch after a long time is converted into glucose, and cane sugar is inverted through the action of the intestinal secretion (Demant[4], Brown and Heron[5], K. B. Lehmann[6], Frick, Turby and Manning[7], and Miura[8]). Similarly F. Röhmann and Lappe[9] have found that an extract of mucous membrane from the small intestine of the calf or dog can convert milk sugar into glucose. The secretion from Lieberkühn's glands has no action whatever upon albuminoid bodies and fats

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* In diseased conditions, marked odourless eructations may occur some time after eating. It appears very probable to me that this results from decomposition of the hydrochloric acid of the stomach by the alkaline intestinal secretion.

(Frick[10], Ellenberger and Hofmeister[11], K. B. Lehmann[6], Wenz[12], and others). The secretion from Lieberkühn's glands in the large intestine appears to be mostly mucous.

2. The Pancreatic Juice

The action of the pancreatic secretion has been studied principally upon animals in whom fistulæ have been made. A few observations have been made with the secretion from fistulæ following the extirpation of pancreatic tumours, particularly cysts. I have myself made a few such observations. The credit for most of our knowledge of the function of the pancreas is due to Cl. Bernard, C. Ludwig, Bidder and Schmidt, Heidenhain and Bernstein. The fundamental studies of v. Mering and Minkowski first brought out the importance of the pancreas in diabetes.

In experiments upon animals two varieties of fistulæ, the temporary and the permanent, are employed. If a fistula be made in a dog after a hearty meal, the secretion which flows will be profuse, viscid, and very active. Activity ceases, however, after a few days—sometimes after a few hours—and it is succeeded by a thin, watery, inert secretion, very poor in albuminoids, and resulting very probably from inflammatory changes. In a few cases it has been possible to obtain an active secretion from a permanent fistula. It is therefore easily understood that analyses of the secretion vary very much.

The normal secretion of the pancreas is a clear, colourless, and odourless, viscid fluid, of a strongly alkaline reaction. The alkalinity is due to sodium carbonate which is present in from 0.2 to 0.4 per cent. The secretion is highly albuminous and coagulates strongly upon boiling. In addition to the above, it contains leucin, fats, fatty soaps, and the following mineral ingredients: Alkaline chlorids and carbonates, phosphoric acid, and some calcium, magnesium, and iron.

In the secretion from a temporary fistula of a dog, C. Schmidt[13] once found 9.92 per cent of solids, and at another time 11.56 per cent; while Zawadsky[14], in examination of pancreas fistula of a man, found 13.25 per cent of solids, of which 9.2 per cent were proteids and 0.34 per cent mineral substances. Herter[15] found 24.1 per cent solids in the secretion of a man; this secretion, though very active, seems to have been pathological in other respects. From the above analyses, we may regard 10 per cent as the average amount of solid matter.

The most important pancreatic ferments are the *proteolytic*, the *amylolytic*, and the *fat-splitting*.

(*a*) At the proper temperature the *proteolytic ferment*, trypsin (Kühne), is capable of converting proteids in a very short time into albumoses and peptones. This ferment does not exist preformed within the gland, but is there instead as trypsinogen (Heidenhain and Podolinski[16]). Trypsinogen is changed into trypsin through bacterial influences, contact with air or water, or with dilute acids (e. g., 1 per cent acetic acid). Trypsin is most active in a 0.2- or 0.4-per-cent solution and at 40° C. Small amounts even of free mineral acids retard its action, but organic acids must be present in stronger concentration. The kind of albuminous matter is not without influence. Thus, as in gastric juice, fibrin is digested much quicker than is coagulated egg albumen.

The initial stage of proteid digestion with trypsin differs very much from that with the gastric juice. Whereas in the latter there is a preliminary swelling of the albuminoid substance followed by gradual solution, in trypsin digestion there is a breaking up, or, more properly speaking, a melting away.

As in the stomach, so, too, in trypsin digestion the metamorphosis of the albuminoids is not a sudden but a gradual one. According to Kühne, who has studied the proteolysis of trypsin digestion more carefully than any one else, the albuminoids are first converted into deutero-albumoses, but after a very short time peptones are also present. One portion of these peptones—Kühne's *antipeptones*—remains unchanged throughout the entire digestion, even though this may continue for several days, while another portion—Kühne's *hemipeptones*—yields products of decomposition, the most important of which are the crystalline amido-acids—leucin, tyrosin, and aspartic acids. These latter products have been well known for a long time; of late, E. Drechsel and Hedin[17] have succeeded in isolating two additional products of trypsin digestion, lysin and lysatinin. Finally, a chromogen, tryptophan, is regularly formed during tryptic action upon albuminoids[18]. With bromin or chlorin water, or with chlorate of potash solution, tryptophan yields a violet colouring matter, readily absorbed by amylic alcohol.

Trypsin also acts upon the gelatins, causing them to lose their gelatinous qualities and to be converted into gelatin peptones. Kühne's and Roberts's[19] investigations with the trypsin of pigs' pancreas have shown that this ferment curdles milk. My own investigations[20] have proved that milk is not curdled by the biliary juice contained in the duodenum (a mixture of bile, pancreatic juice, and the secretions of Brunner's and Lieberkühn's glands), but that it is immediately peptonized. If the gelatin-yielding substance of connective tissue is previously swelled by the action of acids, or shrunken through the agency of heat, it will be dissolved. Elastic membranes, as well as the membranes of fat globules, are immediately dissolved. Chitin and keratin apparently are not dissolved by trypsin.

(*b*) *Pancreatic Diastase.*—This ferment has the property of converting starch and glycogen into sugar, the end products being mostly maltose, together with small quantities of dextrose (Musculus and Gruber[21] and v. Mering[22]). This conversion is a hydration process, and the intermediate products are the same as those occurring in the diastatic action of ptyalin, with which ferment pancreas diastase is in all probability identical. Taken in their order of occurrence these intermediate products are: first, soluble starch, which still gives a blue colour with Lugol's solution of iodin; next, erythrodextrin, which gives a violet colour; and, finally, one or more of the achroodextrins, which remain uncoloured upon addition of Lugol's solution.

In addition to diastase, pancreatic juice also contains invertin, although in much smaller quantities than does the intestinal secretion. It is assumed that this ferment gradually completely converts maltose into dextrose (glucose). In like manner cane sugar and milk sugar are also converted into glucose by invertin. Glycogen is first converted into a maltoselike sugar, and then gradually also changed into glucose.

(*c*) *The Fat-splitting Ferment* (*Steapsin*).—This ferment splits up fats into fatty acids and glycerin. In the presence of alkalies these fatty acids combine to form soaps; furthermore, they aid very much in the emulsification of fats. We shall return to this when we come to speak of bile. Steapsin has not yet been isolated, and the attempt has therefore been made to refer the splitting up of fats in the intestines to the action of bacteria. The researches of Nencki[23] have, however, shown the positive existence of a fat-splitting ferment in pancreatic juice. In my own investigations[24] with the small intestinal juices of man I have observed marked fermentative dissolution of fat.

According to Nencki and Baas[25], pancreatic ferment decomposes other esters besides the neutral fats. We shall speak of the part played by the pancreatic juice during digestion when discussing intestinal digestion in its entirety.

3. The Bile

The importance of the bile in digestion was formerly very much overestimated. At the present day bile may be looked upon as possessing more of an excretory than a secretory importance, since it has to render innocuous the products of body metabolism stored up in the liver and safely conduct them from the body by way of the intestines. It would be going too far, however, to assign but this one function to the bile. Without doubt it has an important part in intestinal digestion, especially in regard to the fats. This will be referred to again later.

The composition of bile varies with different animals; that of the dead body is different from that of the living, and that flowing from the biliary passages is not the same as that contained within the gall bladder.

Human bile, such as is obtained immediately after death from executed persons, is usually of a golden-yellow colour with a shade of brown. Greenish-coloured bile has also been seen, especially during operations. Bile such as I have seen regurgitated into the stomach in numerous cases of deeply situated duodenal stenoses was almost always green; in a very few cases where there was a normal condition of the duodenum, I saw bile of a golden-yellow colour. The taste of bile varies in different animals; that of man and of cattle is bitter and has a sweetish after taste, while that of the rabbit and the pig has a purely bitter taste. The specific gravity of that in the gall bladder varies from 1001 to 1004. The reaction is faintly alkaline, due to the presence of the carbonate of soda (.02 per cent) and of alkaline sodium phosphate. The amount of solids in bile obtained from fistulæ varies so largely that figures are valueless. This holds true, too, for the total quantity in twenty-four hours of bile from fistulæ, for an appreciable but immeasurable portion must flow into the small intestine itself. Besides, as Stadelmann has correctly shown, marked variations in the biliary secretion may occur under normal conditions. Bile contains very much mucin as well as mucoid nucleo-albumin.

The most important constituents of bile are the two biliary acids and their salts—the glycocholate of soda and the taurocholate of soda—cholesterin, and a number of biliary colouring matters. In addition there are small quantities of lecithin, neutral fats, soaps, urea, and mineral salts.

As regards the two biliary acids, it should be stated that glycocholic acid ($C_{26}H_{43}NO_6$), which is combined with sodium and traces of potassium, is more abundant in man; while taurocholic acid ($C_{26}H_{45}NSO_7$) is found in carnivora and in goats and sheep. As can be seen from the formula, taurocholic acid contains both nitrogen and sulphur, whereas neither of these substances is present in glycocholic acid. Both acids have a bitter taste and are dextrogyrous; they are decomposed upon boiling with caustic potash, glycocholic acid splitting up into glycin and cholic acid, and taurocholic acid into taurin and cholic acid.

The biliary colouring matters met with under normal conditions are bilirubin (reddish yellow) and biliverdin (green), and one colouring substance closely related to hydrobilirubin (urobilin).

Bilirubin (identical with hæmatoidin) is found in the golden-yellow bile of man, and is present to a marked degree in biliary concretions as bilirubin-calcium. It is insoluble in water, sparingly soluble in ether, a little more so in alcohol, and readily in chloroform, especially if warmed. Upon evaporation it may be obtained partially in crystalline form, partially as an amorphous substance. Solutions of bilirubin in chloroform yield reddish-yellow rhombic scales which are exactly like hæmatoidin. For other properties of bilirubin, as well as for its chemical reactions, we refer the reader to works on chemical physiology.

Biliverdin results from oxidation of bilirubin, and is met with in the bile of man, of the lower animals, and also in the urine of jaundice, and to a less extent, too, in gallstones. It is an amorphous substance readily soluble in alcohol, sparingly in ether, and insoluble in chloroform, thus differing widely from bilirubin.

Hydrobilirubin results from the reduction of bilirubin, and is constantly present as colouring matter in normal fæces. It is in all probability identical with urobilin and the so-called stercobilin.

Cholesterin is held in solution by the bile salts. It crystallizes in colourless rhombic plates, is insoluble in water, but soluble in hot alcohol, in ether and chloroform.

It is claimed that in addition to the above-described constituents, there is also a diastatic ferment present in bile; but very probably it is the diastase of the salivary and intestinal secretions.

Importance of Bile in Intestinal Digestion.—Apparently the principal function of the bile is to promote the absorption of fat. This is readily seen from the fact that dogs with biliary fistulæ absorb far less fat than do normal animals. Whereas a normal dog, according to Voit[26], given 150 to 250 grams of fat will absorb

99 per cent of it, one with a biliary fistula given 100 to 150 grams can absorb but 40 per cent, the remaining 60 per cent passing off with the fæces. Röhmann[27] arrived at similar conclusions. On the other hand, it may be stated as an actual fact that no disturbance of digestion occurs when the bile is conducted externally, provided, however, that the amount of fat partaken of be not too large.* There is still much doubt as to the manner in which bile promotes fat absorption. We know that it favours the emulsification of fats. This action, according to Neumeister[28], results from its cholates, which dissolve the potassium and magnesium soaps that are insoluble in the other fluids of the small intestine. According to the more recent views of physiologists, especially those of Heidenhain[29], bile promotes the entrance of fat into the epithelial cells by favouring fat emulsification, and by imparting to the surface of the cells a capillarity toward fat. Further characteristics ascribed to bile are antiputrefactive qualities and stimulation of intestinal motions (dogs with biliary fistulæ and human beings with occlusion of the bile passages are obstinately constipated).† The latter results from the free bile acids which normally are always present in the small intestine. The fæces of dogs with biliary fistulæ have a most decided stench, resulting from the decomposition of carbohydrates. Bile has no digestive action upon albuminoid bodies.

INTESTINAL PERISTALSIS

(*Motus peristalticus*)

The object of intestinal peristalsis is to secure a mingling of the contents and their onward propulsion. Thus not only is the pabulum brought into intimate contact with the digestive juices, but as soon as this is accomplished the contents are further conducted through successive portions of the intestines, and thereby thorough absorption is secured. From this it follows that the small intestines are mostly concerned with peristalsis, and that peristalsis takes place almost exclusively during the act of digestion.

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* Compare, for example, Mayo Robson, Proc. Roy. Soc., vol. xlvii, 1890, pp. 499–524; Copemann and Winston, Jour. of Physiology, vol. x, 1889, pp. 213–231; Noel Paton and John Balfour, Laboratory Reports issued by the Royal College of Physicians, Edinburgh, 1891, vol. iii, pp. 191–240 (cited from Gamgee, Physiologische Chemie der Verdauung, 1897, p. 288).

† As far as man is concerned, I can not, in general, personally confirm this rule, which we find laid down with the greatest certainty in almost all text-books on physiology.

The excellent investigations of Braam-Houkgeest[30], of Sanders-Ezn, and of Nothnagel[31], who studied peristaltic movements in animals placed in physiological salt solution, have acquainted us fully with all the details. We may distinguish three types of normal intestinal movements: peristalsis proper, pendulum motion, and rolling motion.

1. *In peristalsis proper* there is a widening of the lumen of the gut, followed by a narrowing—a wavelike motion, which passes with moderate speed over a certain length of intestine toward the anus, and thereby causes a visible onward propulsion of intestinal contents. In the colon the peristaltic wave is represented by depressions and prominences of the haustra following each other in regular succession.

2. *Pendular motions* are always limited to a short portion of the gut. In these the gut is moved to and fro without any perceptible change in the width of its lumen. There is no propulsion of the contents, but, instead, a mingling of the same. Propulsion takes place only where peristalsis is associated with pendular motions, a combination which has been observed. According to Nothnagel, pendular motions may be seen particularly in the middle and lower segments of the intestines when these are filled with pabulum. The motions may last as long as fifteen minutes, when they suddenly cease, to begin again after a long time (one hour to an hour and a half) without any special cause. This period of cessation is said to be shorter in the lower portion of the ileum only. Braam-Houkgeest observed pendular motion in empty as well as filled intestines; Nothnagel in the latter only.

3. *Rolling motions* occur where a segment of the gut is strongly distended by gas and at the same time has fluid contents. Such a segment is from 10 to 20 centimetres long. Its contents are propelled with a rapidity closely resembling a condition known as violent peristalsis. The following is Nothnagel's classical description:

"A circular constriction constantly progresses behind the distended intestinal coils, causing such a propulsion of the fluid and gaseous contents toward the cæcum that the widely distended intestinal parts roll about like a wheel quickly revolving in water. And now a surprising occurrence is often observed: without the slightest external cause, the peristalsis may suddenly cease at any segment whatever, to begin very strongly again after an indefinite interval."

An analogy to these movements is furnished us by the familiar violent peristalsis (*tormina intestinorum*) often experienced very soon after a serious dietetic error. But, as Nothnagel distinctly emphasizes, these movements do not involve the entire length of the small intestine; they completely cease after rapidly passing along a limited portion, and suddenly and without apparent cause begin anew in another portion. They are most active in the duodenum and jejunum; movements in the duodenum being excited by the bile and pancreatic secretion. Farther down in the small intestine the peristaltic waves are less frequent and slower.

In consequence of the peristaltic action of the small intestine, the pabulum from a large meal passes in from two and a half to three hours from the pylorus to the ileocæcal valve; whereas, owing to slowness of peristalsis in the large intestines, food requires at least twelve hours to pass through the much shorter distance between the cæcum and the rectum.

According to investigations of Braam-Houkgeest, which have been fully verified by Nothnagel, peristaltic movements in normal intestine with free lumen proceed always toward the anus and never toward the pylorus. Nothnagel therefore rejects the occurrence of antiperistalsis, such as has been described by older investigators, particularly Engelmann, as taking place physiologically. There appeared, however, to be two exceptions: When Nothnagel introduced active irritative fluids (e. g., concentrated solutions of sodium chlorid, of potassium nitrate or bromate, or weak solutions of sulphate of copper) into the rectum of rabbits, he observed antiperistaltic movements as far as the cæcum, but these movements could be induced from the rectum alone; they were never evoked by irritative solutions of salt introduced through the stomach. Even after artificially produced acute intestinal obstruction in animals, Nothnagel did not observe antiperistalsis. There was, indeed, a backward movement of the intestinal contents; this resulted, however, not from antiperistalsis, but rather from a procedure which Nothnagel has designated "recoil contraction." In consequence of the lively peristalsis and of the increase in intestinal secretions above the stenosis, there is a large accumulation of the contents in the suprastenotic part. As the wall of the gut becomes distended, the portion immediately above the stenosis contracts strongly; this may result in forcing the contents upward. Nothnagel does not believe that the regurgitation of intestinal contents in intestinal obstruction is brought about in this manner, but maintains that it results from pressure of the abdomen upon the distended paretic wall of intestines above the stenosis.

The above-mentioned investigations of Nothnagel show that no backward peristaltic waves can be observed in the physiologically functionating intestine.

A different condition can be observed when we employ mild irritants, such as injections of normal salt solution, which to a certain extent are still physiological. Thus Grützner[32] has lately called attention to the interesting fact that the injection of physiological salt solution into the rectum of man and the lower animals produces antiperistaltic movements, which involve not only a short segment, but also the entire intestine up to the duodenum; furthermore, Grützner showed hereby that small solid particles (starch granules, hairs, charcoal, etc.) which are suspended in the injected fluids are carried up to the duodenum, or even in part to the stomach, being found in the wash water from the latter organ.

Simultaneously with these antiperistaltic movements, which pass immediately next to the mucous membrane, there also occur peristaltic movements by which fæces and chyme are brought downward.

Thus far control examinations by others do not entirely confirm Grützner's conclusions. Christomanos[33], Dauber[34], and Wendt[35] think positive results were obtained because the animals licked their own intestinal evacuations; where this was carefully guarded against, negative results were obtained. By experiments upon human subjects, Swieżynski[36] was able to demonstrate that lycopodium introduced into the rectum passed upward into the stomach. He, however, was unable to determine with certainty the part which sodium chlorid played in this experiment. Be this as it may, the investigations of Grützner do not prove the occurrence of a true antiperistalsis, but only show that food stuffs introduced into the rectum may pass upward within the intestine. We fully coincide with Riegel[37], who regards the superficial epithelium of the intestines as the chief causative factor in this upward movement.

Wherein lies the impulse for intestinal movements? Investigations made thus far upon this point show that the intestinal wall has automatic movement centres similar to those of the heart, and, furthermore, that the central nervous system exerts an important influence upon physiological and pathological intestinal movements. The principal seat of the automatic centres is very probably the nerve and ganglion plexuses of Meissner and of Auerbach. Nothnagel could elicit intestinal movements by touching various spots in the intestines with a sodium or a potassium salt, and thus proved beyond all doubt the presence of nerve centres within the wall of the intestines.

The relations between the cerebro-spinal nervous system and intestinal movements are very complicated. The pneumogastric undoubtedly plays a certain part here, for irritation of that nerve causes or increases a movement throughout the entire small, and the upper portion of the large, intestine. The entire large intestine, according to the latest researches of Pal[38], receives motor fibres from the pneumogastric. We know from Pflüger's celebrated experiments that the splanchnic nerve inhibits intestinal peristalsis. This inhibition may be direct, or it may be brought about indirectly by the anæmia which results from irritations of the splanchnic. According to the most recent experiments of S. Mayer and von Basch, the splanchnic nerve exerts an inhibitory influence only as long as the blood in the capillaries is not venous; when the blood becomes venous, then stimulation of this nerve causes increased peristalsis. Investigations of Nasse show that the splanchnic also contains direct motor fibres, but Pal[39] concludes from his experiments that the action obtained is a reflex one. In like manner, stimulation of the cortex of the brain and of the optic thalamus may inhibit or accelerate intestinal movements: the acceleratory stimulus passes through the pneumogastric, the inhibitory through the spinal cord (Bechterew and Mislowski[40]).

J. Pal[38] has furthermore shown that the splanchnic centre is not the only inhibitory one of the small intestines, but that there are others lower down in the spinal cord.

Contrary to the small intestines and the upper part of the large, the lower part of the large intestine and the rectum receive inhibitory as well as acceleratory fibres from the sympathetic.

In connection with innervation of the rectum, Fellner[41] has called attention to a remarkable antagonism existing between the nervi erigentes and the nervi hypogastrici of the dog. Whereas the former nerves contain motor fibres for the longitudinal and inhibitory for the circular muscles, the reverse is true of the hypogastric. L. Exner[42] disputes the evidences lent by Fellner's experiments, but they have recently been confirmed by Pal[43].

Normal peristalsis may be increased or diminished through external as well as internal agencies. The external agencies include cold, massage, and the faradic current; the internal, mechanical, chemical and thermal, vaso-motor and central stimulation. Those mechanical agencies which cause an increase in normal peristaltic movement are foreign bodies, stenoses, parasites, and particularly indigestible foods; the chemical agencies include laxatives and tainted food stuffs, with their accompanying products of decomposition. The thermic excitants of peristalsis are cold, especially in the form of cold drinks, ice, or of cold injections. Circulatory disturbances resulting from congestion of the portal system may cause an increase or a slowing of peristalsis. Finally, we may have increased peristalsis resulting from certain conditions of the central nervous system—e. g., crises entériques of tabes, tormina ventriculi nervosa (Kussmaul), in neurasthenics, hysterics, etc.

Conversely, similar conditions may cause the peristalsis to diminish, or to cease entirely. Mechanical causes are very frequently found in women; retroflexion of the uterus is one of the best known, although not the only example.

As examples of thermal sedatives, we have warm, moist applications (cataplasma) and warm enemata. Where we have increased intestinal movements, the diet employed may, from its nature, act as a chemical sedative—e. g., gruels in diarrhœas. The narcotic drugs (opium and its derivatives, belladonna, etc.) should be classed with those chemical agents which diminish intestinal peristalsis. The latter may also be indirectly influenced by remedies which tend to diminish intestinal secretion (the so-called astringents).

In the course of many diseases of the intestinal mucous membrane there is a gradual decrease in normal intestinal excitability. In intestinal stenosis we may have a temporary or a permanent paralysis, resulting from paretic effects of fluid or gaseous products. Disturbances of intestinal circulation (hæmorrhoids, congestion accompanying uncompensated valvular affections, etc.) frequently cause a decided slowing of peristalsis. Furthermore, central nervous impulses may not only accelerate, but may weaken or even inhibit intestinal movements—e. g., meningitis, cerebral tumours, etc. In functional neuroses (neurasthenia, hypochondriasis, and hysteria) there may be a marked diminution in the peristaltic movements. Lastly, feeble peristalsis has been observed as a congenital inherited condition.

INTESTINAL ABSORPTION

The intestinal canal is the principal seat of food absorption. As has been proved by the most recent researches of von Mering, Moritz, Hirsch, and Brandt, the absorption of substances (even in solution) from the stomach is very limited. It has been demonstrated that besides water, the intestines also absorb peptones (or albumoses) as well as the various saccharins, salt solutions, and fats, and give up these substances to the lymph and blood channels.

Formerly absorption was regarded as a purely physical process—for the most part one of simple diffusion. But since colloids (albumins and gelatins) have very poor osmotic properties, this explanation would apply to crystalloid substances only (salt and sugar). Furthermore, this theory does not account for the entrance of the fats into the chyle. The view which is now accepted is that absorption of food particles results mainly from a specific function of the living protoplasm. This theory has sprung up within recent years, and has been evolved from the studies of Hoppe-Seyler, Heidenhain, I. Munk, and others.

We shall now briefly describe the manner in which absorption takes place in the intestines. First, as regards the absorption of peptones. It is well known that peptones do not enter the blood current as such, but in the form of albuminoids. This change is essential, for it has been proved that peptones injected into the blood are quickly excreted by the urine (Plósz and Gyergyay[44], Hofmeister[45]). Consequently peptones must be converted into albuminoid bodies before they enter the blood. Opinion is divided among physiologists as to where this conversion takes place. Thus Hofmeister[46] ascribes the greater importance in assimilation and absorption of the proteids to the leucocytes found so plentifully upon the surface of the intestine during digestion; whereas Heidenhain[29] credits the leucocytes with but a very minor part in the reconversion of albuminoids from peptones, and ascribes this rather to the epithelial layer of the villi.

It is important to inquire into the utilization of albuminoids where pancreatic juice is entirely absent. After complete extirpation of the pancreas in dogs, Abelmann[47] and Minkowski found an average utilization of 44 per cent of albumin ingested, while, after partial extirpation, 54 per cent was utilized. Similar experiments of Sandmeyer[48] showed a utilization of from 62 per cent to 70 per cent, which was markedly increased by the administration of raw beef pancreas. In the latter instance, figures coinciding fully with those from normal animals were obtained.

As regards absorption of the carbohydrates, that of glucose levulose and galactose is direct, while maltose and cane sugar are first converted into glucose within the intestines, and as such are then absorbed. Experiments of Ludwig and von Mering[49] upon lower animals, and of I. Munk and Rosenstein[50] upon man, tend to show that carbohydrates are not absorbed through the chyle vessels, but pass directly into the blood. Should there be an excess of blood in the intestine, some of the sugar will pass directly into the chyle vessels and into the thoracic duct (Ginsberg[51]). It appears from the investigations of Hoppe-Seyler[2], von Mering[49], and Otto[52], that cane sugar as well as dextrinlike substances may be found in the blood, though only in small quantities.

There is also a limit to the amount of sugar which can be conveyed to the blood-vessels of the liver; when this limit is exceeded, the excess in all probability passes directly into the lymphatic system and is not brought to the liver at all. A portion of it readily appears in the urine (alimentary glycosuria). The so-called "limit of assimilation" varies with the individual person as well as with the variety of sugar.* Sugar is stored up in the liver and to a less extent in the muscles and glands, in the form of glycogen, a non-oxidizable substance. The pancreas is the main though not the only factor in the digestion and absorption of sugars. Although Minkowski and Abelmann[47] found that only 57 to 61 per cent of starches were absorbed after total extirpation of the pancreas in dogs, the experiments of Fr. Müller[53] on man have nevertheless shown that, even with total occlusion of the pancreatic duct, starches are absorbed with hardly any loss whatever.

Fats are absorbed in the intestines after previous emulsification by the pancreatic secretion and the bile. Owing to the great abundance of alkalies present, the fatty acids are converted into soaps. Fats are very probably absorbed through the lymphatics. This has been proved by the investigations of Munk and Rosenstein[50] in their case of lymphatic fistula. In their experiments they found 60 per cent of the fats originally given in the chyle, and only 4 to 5 per cent of that amount was saponified. Furthermore, upon feeding with erucic acid, a fat entirely foreign to the body, they again obtained 37 per cent, and this in the form of neutral fat. It follows from this that the fatty acids do not enter the lacteals in their original form, but as neutral fats. I. Munk assumes also that fatty soaps are for the greatest part converted into neutral fats before entering the lymphatics.

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* Compare von Noorden, Pathologie des Stoffwechsels, Berlin, 1893.

To sum up, absorption of the fatty portion of the food takes place as follows: 1. Finely emulsified fat, as such, is either taken up by the migratory lymph cells lying upon the free surface of the intestinal wall (Zawarykin[54] and Wiedersheim[55]), or else it passes directly into the villi, entering between the spaces at the base of the cylindrical epithelia (Heidenhain). 2. Fatty acids are converted into neutral fats, and are likewise taken up by the lymphatics. 3. Fatty soaps are reconverted into neutral fats and taken up by the lymphatics. Since fatty acids, as such, are not found again in the chyle, we must assume with I. Munk[56] and von Walther[57] that they are converted within the intestinal wall into neutral fats by the addition of glycerin, or even upon the intestinal surface itself (von Walther).

As regards the *soaps*, it would appear as though they could combine with glycerin to form neutral fats within the parenchyma of the villi. We must, however, admit the impossibility of explaining the source of the glycerin necessary for this change. Possibly it may be a portion of that made free in the splitting up of fat by the pancreatic secretion; at all events, the investigations of Perewozisnkoff[58], and the later ones of Will[59] and C. A. Ewald[60], have shown that the living intestine is capable of forming neutral fats when the component parts, fatty acids and glycerin, or fatty soaps and glycerin, are introduced into it. Neumeister[28] quite correctly points out that the epithelial cells play an important part in this conversion.

Variety and constituency of the fats are of great importance in determining their digestibility. I. Munk[61] and Arnschink[62] have shown that fats with a high melting point (mutton tallow, stearin, etc.) are not so completely absorbed as the more easily melted fats (lard, goose fat, olive oil, butter, etc.). According to Munk and Rosenstein[50], a firm fat like mutton tallow is more slowly absorbed than a fluid like lipanin. Besides this, a free fat is more readily absorbed than one which, like lard, is inclosed in an envelope (Rubner[63]). I find interesting the fact mentioned by Fleischer[64], that Hippocrates had already referred the difficulty in the digestibility of eels to the stearin contained in them.

How does occlusion of the pancreatic duct or extirpation of the pancreas affect fat digestion? The absence of pancreatic juice is certainly of great importance in this connection. According to Minkowski and Abelmann[47], all fats (neutral as well as mixtures of fatty soaps and fatty acids) fed to dogs under these conditions appear in the fæces. Milk forms the only exception; over half of its fat is absorbed. An increase in the absorption of fat was observed when pancreas of beef or pig was added to the food. Other experiments upon the absorption of fat, especially in man, contrast with the results just cited. Thus Sandmeyer[48] found great variations in the absorption of unemulsified fats in his dogs (0–78 per cent). Where emulsified fats in the form of milk were given, 42 per cent was absorbed. Teichmann[65] found no alteration in fat absorption in rabbits in whom the pancreatic duct had been tied. Fr. Müller[53] found rather abundant fat absorption in a case of pancreatic fistula consequent upon the extirpation of a cyst.

Though the results just enumerated are so very contradictory, it is not incorrect to assume that disturbances in the assimilation of fat arise wherever there is a long-continued exclusion of pancreatic juices, and (as is usually the case in man) also of the bile. From this it by no means follows that these secretions are indispensable to the support of life, for there are apparently vicarious forces which to a certain extent can offset the disturbances occasioned by the absence of these digestive secretions.

Absorption from the large intestine is relatively unimportant. However, when the activity of the upper portions of the absorptive system is in abeyance, the large intestine may for a time act vicariously. This we learn from the employment of rectal nutrition. Water, especially, is very readily absorbed from the large intestine. As proved by Eichhorst[66], Voit and Bauer[67], Leube[68], Ewald[69], Huber[70], and Kohlenberger[71], albuminoids and albumoses are also absorbed from the rectum and the large intestine. My own experience with milk has taught me that it is almost entirely absorbed from the lower segment of the large intestine. Starch which has not been converted into sugar is slowly acted upon by the bacterial ferments, and ultimately decomposed into lactic acid, butyric acid, etc.

Fats, especially emulsified fats, are slowly absorbed from the large intestine (Kobert[72], Munk and Rosenstein[50], P. Deucher[73]). The amount taken up is, however, comparatively slight, only 10 per cent of that present in a rectal injection being absorbed (Deucher). The absorption of oils may be increased by the addition of normal salt solution.

The immediate result of a liberal absorption of food constituents from the intestines is a gradual increase in the consistency of the residue, which slowly assumes the form of normal fæces. At the same time the acid reaction of the masses becomes gradually neutral or feebly alkaline.

THE EXCRETORY FUNCTION OF THE INTESTINAL CANAL

Besides absorption and secretion, the intestinal canal, like the stomach, has also the function of excretion. It may thus rid itself of superfluous substances as well as of waste materials from the blood, all of which it excretes with the fæces. This function of the intestines, which has been closely studied within the last few years, is also of clinical interest, and therefore deserves brief mention.

We know from Hermann's experiments[74] with animals, but even more so from Fr. Müller's experiments[75], in connection with Senator, Lehmann, I. Munk, and Salkowski, upon the two professional fasters Cetti and Breithaupt, that during fasting the fæces contain a number of organic and inorganic substances, a portion of which, at least, must be regarded as excreted matter. Kobert[72] deserves credit for having made similar studies upon the isolated large intestine of men, and for having thus determined the share taken in excretion by that portion of the intestinal canal. He found that in twenty-four hours an inactive large intestine excreted an average of 0.9684 grammes dry substance, the percentage of whose ingredients varied from 3.35 per cent inorganic and 96.65 per cent organic ingredients, to 57.52 per cent inorganic and 42.48 per cent organic substances. The inorganic ingredients were sodium, calcium, magnesium, iron, and phosphoric, sulphuric, and hydrochloric acids; the organic, mucin, albumin, keratin, fatty acids, soaps, and neutral fats. A large proportion (12.793 per cent) of the inorganic substances consisted of calcium and of phosphoric acid (44.52 per cent). This substantiates the results of Fr. Müller's experiments on dogs, as well as those of von Noorden and Belgardt[76] on man, in which they found large amounts of calcium and phosphates in the excretions, particularly in those from the large intestine. From investigations in a case of fistula of the ileum, Honigman[77] recently arrived at the same conclusions. Particular interest lends itself to the discovery made by Kobert, that the large intestine can also excrete fat. He found that the total amount varies from 9.32 per cent to 6.48 per cent of the dried substance; 90 per cent of this total amount being fatty acids, 9 per cent neutral fats, and the remainder fatty soaps. From this it may be seen that intestinal excretion constitutes an important auxiliary to the functions of the kidney. The occurrence of profuse diarrhœas in extensive parenchymatous changes of the kidneys is in all probability an attempt on the part of the organism to vicariously rid itself of waste material through the intestinal canal. From investigations thus far made, we would regard the large intestine as most actively participating in this vicarious action.

THE NATURE AND COMPOSITION OF THE FÆCES

The fæces are made up partly of food which is either indigestible or has not been acted upon by the digestive juices, partly of the secondary products of digestion, and lastly from the remnants of the secretions and excretions of the digestive tract itself. In this latter connection it should be remarked that the empty intestine may produce fæces through its secretions and its exfoliated epithelium (so-called *Ringkoth* of L. Hermann[74]).

The composition of the fæces naturally varies very much according to the nature and range of the diet. Under a vegetable diet the fæcal masses are much bulkier than under an animal one. As an example of this, Voit[78] states that the excrements of a man under a mixed diet amounted to 120–150 grams, with 30–37 grams solids in twenty-four hours; while those from a vegetarian equalled 333 grams, with 75 grams solids. The colour of the fæces is to a certain degree also dependent upon the diet. Under mixed diet it is dark brown; under milk diet, brownish yellow; and brownish black, or even deep black, under meat diet.

Among the food ingredients found in normal fæces are muscle fibres, connective tissue, casein particles, starch fragments, fat, vegetable remnants, horny substances, nuclein, etc. The intestinal mucous membrane and its secretions contribute mucin, cholic acid, dysalin, and cholesterin. The products of intestinal putrefaction found in the fæces include skatol, indol, volatile fatty acids (acetic acid is said to be constantly present in fæces), calcium, and magnesium soaps. Of the inorganic salts in the fæces, the readily soluble alkaline chlorids occur but rarely, while the insoluble combinations —ammonium magnesium phosphates, calcium carbonate, neutral calcium phosphate, and magnesium phosphate—are, on the contrary, very frequently met with, being for the most part derived from the food.

Bacteria and other micro-organisms occur in large numbers in the fæces; according to Woodward[79], they constitute a very considerable portion of the fæces. The only bile pigment found under normal conditions is urobilin (stercobilin); the occurrence of bilirubin or biliverdin is pathologic.

Careful quantitative analyses of the fæces have often been made, but, as might have been supposed, they have given widely varying results. We would therefore desist from mentioning them.

INTESTINAL DIGESTION IN ITS ENTIRETY

In attempting a description of intestinal digestion *in toto*, we encounter serious obstacles; for our knowledge of this subject includes a number of isolated processes which can not be grouped together without the aid of some hypotheses.

What impulse excites intestinal secretion? The opening of the pylorus acting reflexly very likely causes an increased secretion of bile and of pancreatic juice. Concerning the pancreatic secretion, we know from the experiments of Heidenhain[80] and Bernstein[81] that it begins to flow simultaneously with the ingestion of food, and reaches its maximum in from two to three hours. Thereupon the amount declines till the fifth to the seventh hour, increasing anew from the ninth to the eleventh, and then gradually declining again from the seventeenth to the twenty-fourth hour, when it finally ceases.

As regards the bile, we know that it decreases during fasting, and is secreted again after ingestion of food. According to Heidenhain, two maxima in rapidity of its secretion are observed in dogs: the first from three to five hours after food ingestion, and the second from thirteen to fifteen hours. Investigations of Rossbach[82] yielded similar results. In all probability, therefore, the chyme meets with an active digestive juice when it enters the intestinal canal.

The action of the intestinal juices upon the products of stomach digestion is still a matter of controversy. Physiologists, particularly Kühne, ascribe to bile the property of precipitating and destroying not only the albumin and gelatin, but also the pepsin of the stomach contents. From this it would seem as though the precipitation of the pepsin within the small intestines was of extreme importance for digestion. However, my own investigations[20] made with the pure mixed secretions from the intestines of man (i. e., mixtures of bile, pancreatic juice, and probably also the secretion from the glands of Lieberkühn) have shown that these views are incorrect. What does occur is rather as follows: The first faintly acid portions of the chyme occasion alterations in the intestinal juices only in so far as they acidify these to a slight degree, whereby, as we have already seen (see page 27), no destruction of the active intestinal ferments occurs. It is true that with the entrance of strongly acid chyme into the small intestine a precipitation of the albuminoids—but not of the gastric ferments —follows. However, a mixture of duodenal juices and stomach contents with the latter in such proportion that free hydrochloric acid is present exhibits solely and distinctly the characteristics of a pepsin-hydrochloric-acid solution. Rennet ferment also is preserved intact in such a mixture. If we alkalinize such a mixture with dilute soda solution, we may—for a short time at least—be able to observe tryptic action. Later this action can not be brought out because the trypsin is destroyed by the continued action of the gastric acid. It is therefore very probable that in the first stages of intestinal digestion there is simply a continuation of the gastric digestion. Gradually, however, as the intestinal juices increase and the amount of strongly acid stomach chyme poured into the duodenum diminishes, pepsin digestion gives way to that of trypsin. My observations have recently, in great part, been confirmed by Fleischer[64] and Meltzer.

In spite of the differences of opinion between physiologist and clinician, the above observations show that the reaction of the contents of the small intestines varies according to the stage of digestion. Even in the lowermost portion of the small intestines—as Nencki, Macfadyen and Sieber[83] found in a fistula in the lowest portion of the ileum—the reaction of the contents was acid, the acidity averaging one *pro mille* (basis of acetic acid).

As soon as the intestinal ferments can act with full force the digestion of food takes place, the individual ferments acting as already described. Proteids which have not yet been converted into albumoses are peptonized, with the additional formation of leucin, tyrosin, and of aspartic acid; unconverted carbohydrates are at first altered into maltose and (a little) glucose, and finally completely into glucose; fats are split up into fatty acids and glycerin, and are in part emulsified—i. e., brought into an absorbable condition.

In addition to these fermentative changes there are also bacteriological changes which to a certain extent affect the proteids and gelatinous substances, but to a far greater degree the carbohydrates. Their existence has been proved by the above-mentioned instructive investigations of Nencki, Macfadyen and Sieber[83] upon their case of fistula of the ileum. The chyme was of a yellowish or yellowish-brown colour and had an acid reaction. As a rule, excepting for a somewhat burnt smell reminding one of the volatile fatty acids, or still more rarely excepting for an odour of decomposition very like indol, this yellowish mass was *entirely odourless*. Besides acetic acid, fermentation as well as musclelactic acid, volatile, fatty, succinic, and bile acids were also present. In large quantities of this chyme it was impossible either through sense of smell or through chemical examination to detect the merest traces of the characteristic decomposition products, such as indol, skatol, phenol, methyl mercaptan, or of their combinations, phenylpropionic acid, paraoxyphenyl-propionic acid, and skatolacetic acid.

Without doubt the real seat of intestinal putrefaction is in the large intestine. The rôle in food digestion assumed by the large intestine is markedly less than that of the small. The experiments of Nencki, Macfadyen and Sieber showed that 85 per cent of the albumin ingested is digested by and absorbed from the stomach and small intestines, so that but about 15 per cent remains for the large intestines. Undoubtedly digestion does take place in the large intestine, but it is more bacterial than fermentative. Another chief difference from small intestinal digestion is the formation not only of useful products, but also of others harmful to the economy. It is questionable, however, if all bacterial by-products are utterly useless in intestinal digestion—if, for example, certain products are not capable of favourably exciting peristaltic motion.

The best-known decomposition products are those of *albumin*. They have been studied by many investigators, above all by Nencki, Baumann, Brieger and H. and E. Salkowski. The most important are indol, skatol, paracresol, phenyl-propionic acid, phenyl-acetic acid, hydroparakumaric acid, the volatile fatty acids, carbon-dioxide, hydrogen gas, methyl mercaptan, and sulphuretted hydrogen.

Of these, the most important from a practical standpoint, are indol and skatol, because, as has been shown by the investigations of E. Baumann, they combine with the sulphates of the food (previously oxidized to indoxyl and skatoxyl sulphates) to form ethereal sulphuric acids (indoxyl-sulphuric acid and skatoxyl-sulphuric acid), and are excreted as such in the urine. They thus form a gauge (though not a constant one) for the putrefactive processes within the intestines. Phenol passes into the urine as phenol-sulphuric acid; the oxy-acids pass off unaltered with the urine.

The *carbohydrates*, like the albuminoids, are also subject to bacterial decomposition. At the present time we recognise a large number of bacteria, which through fermentative action may on one hand convert starches into sugars, and on the other cause these very sugars to ferment. For example, the bacillus subtilis and the spirillum of cheese are both capable of converting starch into sugar; but further long-continued action of the subtilis on this sugar results, according to van den Velden, in fermentative production of lactic, butyric, and succinic acids. Some bacteria and yeasts can produce invertin, etc.; others, again, such as the bacillus butyricus, convert lactic acid into butyric acid. As final products of carbohydrate fermentation we have a number of gases, the most important of which are carbon dioxid, hydrogen and marsh gas.

Cellulose fermentation results mainly from the action of certain bacteria or vibrios.

Fats are capable of fermentation, especially when in the form of fatty acids, but the special organisms as well as the different steps of the process are entirely unknown to us.

[THE GASES OF THE INTESTINES

The gases occurring within the intestinal tract are derived from three sources:

1. They enter the bowel from the stomach.
2. They pass from the blood into the intestines by diffusion.
3. They are formed within the intestinal canal.

Of these three sources the third is by far the most important and active.

1. The gases that may enter the bowel from the stomach are:

a. Air swallowed during or independently of the ingestion of food (oxygen and nitrogen).

b. Gases contained in food, beverages, and medicated liquids. These consist mainly of carbonic acid, but small quantities of other gases may be introduced with the mineral waters (e. g., sulphuretted hydrogen in sulphur waters, etc.).

c. Gases originating within the stomach. These are partly absorbed, partly escape from the body through eructations, but a certain portion passes into the intestines. In the early stages of normal gastric digestion, micro-organisms swallowed with the food cause some fermentation of the stomach contents with a resulting formation of carbonic-acid gas and hydrogen. According to Miller,[84] this fermentation ceases as soon as a large amount of hydrochloric acid has been produced. Schierbeck[85] has demonstrated that during digestion carbonic-acid gas is produced by the secreting cells of the gastric wall. Strauss[86] and Rosenheim[87] found ammonia present in very small amounts.

Under *pathological* conditions (stagnation with or without the presence of hydrochloric acid, abscess of the wall, ulcerating carcinoma, etc.), in addition to the above-mentioned two gases, hydrogen, sulphuretted hydrogen, ammonia, marsh gas, and other hydrocarbons may be produced. It is but natural to assume that with a patulous pylorus and fair or good gastric motility these abnormal gases may enter the intestines.

2. Bunge[88] states that nitrogen, and Nothnagel[89] that carbonic-acid gas, enter the intestinal canal by diffusion from the blood.

3. Throughout the entire intestinal canal gases are formed from digestive fermentation and bacterial decomposition of the food; in the uppermost portion of the small intestine, from the action of acid gastric chyme and of the free acids of the fats upon the alkaline intestinal and pancreatic juices. The amount and kind of gas formed vary according to the diet and the segment of bowel in question. Gas formation is most rapid wherever active fermentative changes occur—i. e., in the upper segments of the small intestine. Accordingly, less gas is produced in the lower portion of the small intestine and very little in the large bowel. In the large intestine gas formation has almost or entirely ceased, and putrefactive decomposition and inspissation of the fæces take place.

From the decomposition of the alkaline carbonates of the intestinal juices by the gastric chyme and fatty acids in the uppermost portion of the small intestine carbonic-acid gas is formed. After the acids have been neutralized, intestinal fermentation and decomposition begin.

Of the solid food stuffs, the *carbohydrates* yield hydrogen, carbonic-acid gas, and a small quantity of marsh gas, varying proportionately to the digestibility of the carbohydrates. Starchy foods result in the formation of but very little marsh gas, while, as we know from the experiments and investigations of Ruge,[90] Tappeiner,[91] and Planer,[92] those rich in cellulose yield more marsh gas than any other variety of food. Since cellulose is not altered by the gastro-intestinal secretions of man, the formation of marsh gas is no doubt due to bacterial action.

The gases that result from the digestion and decomposition of *proteid* material are formed more slowly than those from the carbohydrates. These are hydrogen, carbonic acid, marsh gas, ammonia, sulphuretted hydrogen, and, according to Lehmann, Hagemann, and Zuntz,[93] nitrogen under certain specific conditions.

As already mentioned, the fats, by the action of their fatty acids upon the alkaline intestinal juices, produce carbonic-acid gas.

Considering the large quantity of gases normally present in the intestine * and the comparatively small amount passed as flatus, their absorption must be a very active one. Regnault and Reisert,[94] Tacke,[95] Zuntz,[93] and others, have demonstrated the presence of marsh gas and other intestinal gases in the expired air.

Under pathological conditions the quantity and composition of gases in a given segment will vary from the normal. It will depend upon the nature of the contents (amount of fermentative material and of fermenting agents—i. e., bacteria, moulds and yeasts), the motility of the bowels, and the condition of the circulation.

In sluggishness or total arrest of the contents (e. g., atony, paralysis, obstruction, occlusion, etc.) there will at first be the products of fermentation, later those of putrefactive decomposition. By his experiments Kader[96] showed that excessive formation of gas within the intestinal lumen was largely dependent upon the circulation in the mesentery. In those experiments in which the mesentery was ligatured, marked meteorism developed; in those in which the gut alone was tied, very little gas developed. (See also page 363 of this work.)

Certain catarrhs, by offering conditions favourable to bacterial and fungoid growth, favour the increase of gases (Nothnagel[76]). The same holds true also for ulcerative and sloughing conditions (carcinoma, dysentery, abscess, gangrene, etc.).

On the other hand, in abnormally rapid peristalsis the contents pass so quickly through the intestine that only easily assimilated food stuffs are decomposed. The starches and sugars and carbonated beverages yield gaseous products within the body; the more resistant carbohydrates, the fats, the proteids, connective tissues, and cellulose pass out very slightly or not at all altered. Hydrogen and carbonic-acid gas are mainly formed; the fæces are often passed in a state of active starchy and saccharin fermentation—Tr.]

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* [A faint idea of the active formation that goes on may be gained by mixing a small amount of fresh fæces (about 5.0 grams) with a little water, placing the mixture in any kind of a fermentation tube, and allowing the tube to remain for a short time in an incubator at 37° C.—Tr.]

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DISEASES OF THE INTESTINES

PART I

GENERAL DIVISION

CHAPTER III

THE HISTORY

In diseases of the intestines the history is of almost greater importance than in gastric affections. In the latter the diseased area is very limited; in the former it is far more extensive. Whereas in gastric disease we can determine the condition through physical and functional examinations alone, in intestinal diseases we are compelled to rely mainly, sometimes even entirely, upon the statements of our patients. Too much care, therefore, can not be expended in obtaining as complete a history as possible.

Naturally here, as elsewhere, our first inquiry should not be concerned with the local disturbances, but with the patient's previous general health. Hereditary tendencies must be taken into account. Our view of a case may be radically influenced by the existence of special dyscrasias, as syphilis, tuberculosis, etc., or by learning of the habitual employment of poisons which we know from experience to be injurious to the gastro-intestinal tract (alcohol, tobacco, lead, mercury, tin, zinc, arsenic, antimony, etc.). Special interest is imparted to a case by a previous history of infectious diseases, particularly such as have local manifestations in the intestines—e. g., typhoid, cholera, dysentery, intestinal tuberculosis, etc. Owing to its etiological relations to cancer, a traumatism received prior to the development of a disease merits special consideration. In the female, the sexual apparatus should receive due attention, since it is frequently a cause of intestinal troubles. We should inquire particularly into the menstrual condition, childbirths, previous operations, as well as regarding any special symptoms the patient may have noticed (leucorrhœa, menorrhagia, metrorrhagia, pains, pressure, etc.). In men, too, there is frequently a direct connection between diseases of the sexual organs and those of the intestines, so that careful inquiry ought never to be omitted. I need but recall diseases of the prostate gland. It is only after having thus obtained a clear and complete oversight of any other local or general affections, which at first seem to have no connection with the present symptoms, that we ought to inquire into the details of the affection in question.

Regarding those points referable to gastric digestion, the reader is referred to Part I (general section) of my work on Diseases of the Stomach. There the most important facts bearing upon intestinal disturbances are discussed.

Whenever there is a suspicion of intestinal disease a most thorough local examination is indispensable. We may begin with the symptoms complained of by the patient, or, preferably, we can pursue a definite plan of inquiry which shall include all the anamnestic data that can be gained from the history of a case.

The following scheme has been of great service to me for many years:

1. PAIN ALONG THE COURSE OF THE INTESTINES

(*a*) Seat of the pain.
(*b*) Origin of the pain; acute or chronic; paroxysmal.
(*c*) Character of the pain; burning, boring, stabbing, tearing, colicky, lancinating.
(*d*) Duration of the pain.
(*e*) Relation of pain to digestion; how influenced, if at all, by quantity and variety of food.
(*f*) Does the attack of pain cease with the passage of wind or of stool; or have these no influence upon the pain?
(*g*) How do rest and motion affect the pain?
(*h*) What is the effect of manual pressure upon the painful area?

As a rule, the statements of the patient regarding the *seat* of the pain are not conclusive for the physician; for it requires very good powers of observation to properly describe to the physician the point of origin of the pain, its extent, etc. Should the pain become intense, the patients naturally think less of its exact situation than they do of its severity. Pain in one region of the intestines alone—that of the vermiform appendix or of the cæcum—is so very characteristic that even the layman can localize it quite well. Its distinct localization indeed differentiates cæcal from similar pains. If the patient tells us that the pain is continuous, that it increases upon motion and diminishes with rest, that the painful area is sensitive to pressure, that the attack began with fever, and perhaps that similar attacks have already occurred, the diagnosis of appendicitis or typhlitis is almost certain. Similarly, at least in well-developed cases of round ulcer of the duodenum, we find a strictly circumscribed area of tenderness.* This pain is characterized by its situation in the prolongation of the right parasternal line somewhat below the gall bladder. Usually the pain begins three or four hours after a meal, and rarely or never radiates toward the back; it is worse after the ingestion of solid food and less after a fluid diet; rest diminishes and bodily movements increase it. If the patient be of the male sex, and his statements coincide with what has just been said, there is at least a well-grounded suspicion of duodenal ulcer, and we should keep this in view in making our examination.

Typical cases with pronounced symptoms, such as are described in text-books, are very rare. These are the classical cases which every physician knows. In the great majority of cases a probable diagnosis of the real condition can only be arrived at after repeated questions as to the location, character, and intensity of the pain. If the patient be seen during his attack of pain, we may sometimes come to a rapid conclusion concerning the nature of the case from the general behaviour, the facial expression, the character of the patient's crying or groaning, etc. For example, the painfully anxious and depressed facial expression of peritonitis, with the increased respirations, the rapid small pulse contrasting with a high temperature, the instinctive dread of the slightest contact and of pressure even of the bedclothes, are so characteristic that an experienced physician, simply from hearing such symptoms described, will at once recognise a serious condition, although he may not be certain as to the exact diagnosis.

The pain accompanying acute or chronic intestinal stenosis or occlusions is not so well characterized. Treves[1] makes the general statement that the pain of complete obstruction is constant (although subject to exacerbations), while that of partial obstruction is intermittent, alternating with intervals of freedom from pain. My own views coincide fully with Treves's, although, as he himself admits, exceptions to the rule occur. In other respects the pain which accompanies intestinal occlusion and stenosis offers nothing diagnostic. From this, however, it should not be inferred that the pain of ileus is like that of volvulus of the sigmoid, or like that of an invagination or even of an obstruction caused by a foreign body, but rather that it is difficult, or even impossible, to make use of these distinctions in the differential diagnosis. We shall return to this point in the special part of this work. Similar difficulties are encountered where there is a history of periodic attacks of pain. If a long interval has elapsed between the time of the last attack and of our examination, the statements of the patient will be uncertain, and for the most part guesswork. In such a case we must seek to establish a connection between the paroxysms of pain and the intestinal functions—viz., to learn if obstinate constipation ordinarily precedes the attack, if the abdomen is full and distended, and, finally, if the passage of gas from above or from below, or of a copious stool, causes the paroxysms to cease. These phenomena very probably point to flatulent colic, although they may also occur with chronic intestinal stenosis. Even in the absence of constipation, more or less severe colicky pains may also be caused by decomposed food which remains for some time within the intestinal canal. Even though we may not at first discover any etiological factor, obstinate constipation complicating intestinal colic should lead us to think of lead colic (colica saturnica). Our inability to establish any connection between the paroxysms of pain and the gastro-intestinal functions by no means justifies our declaring the pains purely "nervous," and thereupon, as careless examiners or novices so frequently do, construing a picture of hysteria or of neurasthenia from answers which the patient makes to leading questions. We should first examine those other organs which frequently cause paroxysms of pain—above all, the liver, kidney, pancreas (?), and the bladder—since stone colics occur in these organs. In women, the uterus, adnexa, etc., must be examined.

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* Round ulcers of the jejunum or the ileum are very rare, and, as a rule, can not be diagnosticated.

One who knows from experience that severe gastralgias and enteralgias may be caused from very small supraumbilical or crural herniæ, and by apparently insignificant præperitoneal lipomata, will be very careful before making the diagnosis of "nervous intestinal pain." At all events, I wish to state here that "enteralgias," as purely functional neuroses, occur much more rarely than, for example, gastralgias. (For further details, see chapter on Intestinal Neuroses in the special part of this work.) It is scarcely necessary to mention that whenever we have a well-grounded suspicion of enteralgia the central nervous system should be most carefully examined (crises entériques of tabes, of myelitis, and of progressive paralysis).

The individual causes which may occasionally give rise to intestinal pain are too numerous to mention. I simply wish to state that we should remember that this pain may be due to swallowed foreign bodies (needles, fish bones), to entozoa,* or more so even to poisonous metabolic products such as occur, for example, after death, or disease of tapeworms and other intestinal parasites. Lastly, we must consider the possibility of nicotin poisoning.

It is of great diagnostic importance to give a careful description of rectal pain. The nature of these pains varies considerably with their cause. When limited to the rectum, the pains may be pressing, boring, burning, or even colicky in character. They may be continuous, or may occur for a short time only, before, after, or during a fæcal evacuation. These rectal pains may be caused by a simple coprostasis in the ampulla recti (the latter sometimes acquiring enormous dimensions). They may be due to hæmorrhoids, to fissures or fistulæ, to rectal ulcers (tuberculosis, fæcal ulcers, degenerated syphiloma), or, finally, to stenoses of the rectum from tumours or cicatricial contractions. It is best not to spend too much time upon the consideration of these different possibilities. In most cases we can at once discover the condition present by digital examination, aided perhaps by further examination with the speculum.

Anal pains may likewise be due to strangulated hæmorrhoids, to fissures of the mucous membrane, to fistulæ, or to periproctitis. Here, too, direct inspection is the surest means of ascertaining the cause of the symptoms.

2. METEORISM, TYMPANITES

(*a*) Acute, chronic, or paroxysmal.

(*b*) Local or general.

Meteorism is a symptom which, when developed to a marked degree, may be complained of by the patient. To be sure, a so-called "meteorism" not infrequently turns out to be an ascites. Meteorism is of diagnostic significance only when taken in connection with the rest of the symptoms. It may be caused solely by the ingestion of food abnormally rich in gases, or which forms gases (carbonated liquids, sauerkraut, the so-called "bloating substances"). The statement that meteorism has suddenly occurred in connection with constipation is important, for this association may speak for a simple coprostasis; we may, however, have to deal with retention of gases in a commencing obstruction, an incarcerated hernia, an invagination—in short, with any condition which can obstruct the downward passage of gases, or even with an acute diffused or circumscribed peritonitis. It may even be one of those singular gaseous distentions occurring in hysterical persons, which may prove a source of great anxiety and of error to the novice.

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* Recently I have observed a case referred to me by Dr. Perel, of Odessa, in which very severe and constant intestinal pains were caused by the tænia nana. As is well known, the pains caused by the tænia solium or mediocanellata are mild, or altogether wanting.

Before beginning the objective examination, close questioning may enable us to form a correct idea of the true condition present.

Chronic meteorism, more or less circumscribed, is also a symptom of manifold significance which can not be correctly valued without thorough investigation. Where the meteorism is circumscribed, we must think of adhesions, or of stenosis, from tumours situated either within the intestines or external to them, and partly connected with other abdominal organs—in women especially with the genital organs.

3. Constipation

(*a*) Acute, chronic, habitual, or periodic.
(*b*) If acute, when was the last movement?
(*c*) If chronic, does it alternate with diarrhœa?
(*d*) Duration of the entire trouble.
(*e*) Are the movements retarded, or spontaneous, or do they occur only after laxatives? If the last, what is the nature of the laxative? Are enemata employed?

Constipation may at times be a harmless condition; at other times it may be extremely severe and dangerous, frequently causing death. The first question which presents itself is: Have we to do with *acute* constipation in a case in which up to the present time there were normal movements? Such a condition may naturally be brought about by various causes—e. g., sudden change in habits of life or in climate, errors in diet, mental excitement, diarrhœas lasting for days and weeks, the administration of opium,* bismuth, tannin, morphin injections, or of other drugs which cause temporary arrest of intestinal peristalsis. Intestinal occlusion or stenosis may be present, or the constipation may be an accompanying symptom of an acute affection of the central nervous system (basilar meningitis) or an acute lead colic. Here, again, only a careful general as well as local examination can demonstrate the true underlying condition.*

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* For a long time I have had under observation a female patient who has suffered from obstinate constipation since an attack of perityphlitis. The cause of her constipation is most probably the use of large doses of opium. (*Cf.* further remarks as to this in the chapter on Perityphlitis, Part II.)

Where we have to deal with a case of chronic habitual constipation, the existence of a functional intestinal weakness (intestinal atony), or of an organic condition, will come into question. This, again, can only be determined by a detailed examination. Where there is constipation lasting for many years and not associated with disturbances of the general bodily conditions, one generally thinks of "functional intestinal weakness" (atony) or of intestinal catarrh. It should never be forgotten, however, that habitual constipation and chronic enteritis also predispose to the development of intestinal cancer with subsequent stenosis (see chapter on Cancer). Even at the present day the view so often expressed, that many years' duration of a disease speaks against cancer, is only correct when greatly modified. The frequent development of cancer upon a coprostasis of many years' duration should teach us to be more cautious. Furthermore, in women we should always examine for some genital disorder as a cause of existing constipation (retroflexed uterus, disease of the ovaries or of the appendages, etc.). Where the entire clinical picture is unlike malignant disease, an exact knowledge of the duration and the previous treatment are important. Constipation is sometimes congenital or inherited (intestinal atony), or it may be acquired during earliest childhood. This is important for the prognosis and the treatment of the case. A knowledge of the nature and effect of the various therapeutic measures employed is essential for the proper appreciation of the diseased condition. *The poorer the reaction of the intestines to laxative measures, the more difficult the treatment, and vice versa.*

Where constipation alternates with diarrhœa, we must first of all decide which is the primary or dominating condition. It is by no means easy for a patient who has doctored much, and who has alternated between laxatives and astringents, to answer this question. To appreciate the real functional disturbance, it is best for the patient to stop all medication for a few days. Should diarrhœa regularly follow constipation, organic stenosis of some kind in some part of the intestines, or else an intestinal catarrh, may be present. Here, again, without a most careful examination, especially of the rectum, it is absolutely impossible to come to a conclusion. Finally, should repeated examinations constantly yield negative results, the question of a nervous enteropathy or of hysteria may, with the greatest reserve, be considered.

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* In this connection it might be remarked that regular movements of the bowels, or even diarrhœas, do not at all exclude intestinal occlusion. Cases with undoubted intestinal obstruction have been described in which fæcal vomiting as well as the passage of wind from below, or even fæcal movements, occurred.

4. DIARRHŒA

(*a*) Acute or chronic; during intervals?
(*b*) How frequently during the day?
(*c*) With or without pain; if present, its character and situation.
(*d*) Does constipation follow the diarrhœa?

A sudden attack of diarrhœa may result from a simple error in diet, or, on the other hand, may initiate a severe, acute, infectious disease (typhoid, dysentery, cholera nostras, and asiatica, ptomain poisoning from decayed meats, etc.). In these cases the disease presents a number of other symptoms, which, together with the objective examination, sooner or later clear up the diagnosis. We may, however, have to deal with one of those frequent cases of infectious enteritis which originate especially during the summer months, from bacterial or other direct local sources (dyspeptic diarrhœa, Nothnagel).

Chronic recurring diarrhœa occurs as a symptom of organic intestinal disease, of local intestinal neuroses (i. e., secretion neuroses, reflex neuroses of tabes and other systemic diseases, crises entériques), or of nervous enteropathies. If the diarrhœa is a symptom of the first-named affections, it may be due to catarrh, atrophy, ulceration, or amyloid disease of the intestinal mucous membrane. In stenoses of the intestine there are generally, every few days, watery, pasty, or partially fluid, partially solid stools. Under predisposing conditions, patients with stenosing, ulcerating carcinoma of the ileum may have continual diarrhœas, usually of a purulent character. I have observed and performed post-mortem examinations on two such cases (for particulars, see Part II of this work). Finally, chronic diarrhœas may occur as a symptom of nephritis (uræmic diarrhœa), of the uric-acid diathesis, of congestion in the portal system, etc. The symptom "diarrhœa," therefore, is so closely connected with apparently widely differing conditions that a satisfactory diagnosis of its cause and nature can not be reached without a careful general and local examination.

5. Character of the Evacuations

(*a*) Consistency, quantity, and colour.

(*b*) Pathological admixtures (mucus, fragments of membrane, blood, pus, tumour detritus, parasites).

(*c*) Odour (feculent or putrid).

In most cases the physician should not be satisfied with the description of the stools as given him by the layman, but should inspect them himself, and eventually examine them under the microscope. If necessary, a chemical examination should also be made. Where the patient's statements are positive, or where they can not be personally controlled, the descriptions as given can not be entirely ignored, for occasionally the statements are of the greatest importance. Sometimes a careful account of the consistency and calibre of the stools is of decisive value in arriving at a diagnosis by exclusion. Thus, persistent cylindrical stools of normal calibre would generally exclude a stenosis of the intestines.

Conversely, stools of an abnormally small calibre do not speak with any degree of certainty for a stenosis of the intestines; in this case the diagnosis must rest upon the presence of other symptoms. The stools may be pasty, semisolid, or fluid in consistency. The first two of these characteristics may be present in the normal individual; the last is always indicative of an abnormal condition and calls for a most careful examination. Scybalous stools indicate a long retention of the fæces in the haustra coli, and this is a frequent cause of attacks of intestinal colic (spastic contraction of the intestines).

The patient's statement of the quantity of fæces passed in twenty-four hours is generally correct. I say generally, because I have often had neurasthenics declare that they have had insufficient evacuations, although they actually passed large quantities of fæces. In many cases the quantity is not commensurate with the amount of food ingested; this may be due to the kind of food taken (especially meat), or to deficient peristalsis. Probably intestinal absorption is increased after a period of fasting, so that even normally in the first few hours after eating again the amount of undigested matter is disproportionate to the amount of food ingested.

The *colour* of the movements may vary considerably from the normal. The patients are most apt to notice the clay-coloured stools that occur with icterus, or sometimes without icterus (see section on Fæces). But since icterus is only a symptom, the diagnosis of the primary trouble is impossible without further knowledge of the cause of the disease.

The pathological admixtures in the fæces which may at times be seen and correctly described by the patients are mucus, mucous membrane, blood, pus, fragments of new growths, undigested food remnants, and parasites.

Where *mucus* is present in large quantities, or is passed alone, it is usually noticed by observant patients. Sometimes these movements of mucous membranes are so characteristically described that the diagnosis—membranous enteritis (colica mucosa)—is readily made. In general the patient's description very seldom yields useful data.

Blood may be mixed with the stools, in a fresh fluid or decomposed state. The latter lends an intensely tarry or pitchy appearance to the stool. However, the description, or even the microscopical appearance, is decisive only in a very few and these otherwise absolutely clear cases (see section on Fæces). It is important here to know if symptoms of severe internal hæmorrhage (collapse, syncope, pallour of the visible mucous membranes, systolic murmur at the apex of the heart, etc.) accompany the intestinal hæmorrhage. The darker the appearance of the blood the more correct are we in assuming that the bleeding is located very high up (stomach or upper portion of small intestines). Tar-coloured blood comes only exceptionally from the lower portions of the intestines. Fluid blood comes mostly (but not always) from the large intestine, including the rectum. By rectal palpation, or by use of the speculum, it can frequently be determined whether the latter is the seat of the bleeding or not. If the rectum can be excluded, various diseased processes may come up for consideration—acute or chronic dysentery, fæcal ulcers, tubercular ulcers, tumours of the large intestine (benign or malignant), and acute or chronic intussusception or other forms of intestinal obstruction.

The admixture of pus with the stools, or the discharge of pus from the rectum, is always a striking symptom. The pus may come from the rectum itself (ulcerating tumours or ulcers, rectal fistulæ), or it may come from intestinal segments above the rectum. We can only determine the seat and cause of the pus formation by means of repeated careful examinations of the entire intestinal canal and of the fæces (*vide* Chapter V).

The fragments of new growths which may be noticed by the patients are broken-off pieces of cancerous tumours (very rare), or exfoliated intestinal polypi. For obvious reasons, it requires a personal examination to pass an opinion upon these. In intussusception, the intussusceptum may become gangrenous and be passed.

Parasites, especially segments of the tapeworm, may be recognised by the layman, but may at times be also confounded with a number of other things.

The importance of the presence of food remnants will be dwelt upon in the chapter on Fæces.

The odour of the normal stools is feculent, but not putrid. Should it be offensive, however, and should the statements of the patients upon this point be very positive, a personal examination of the stools and of the intestinal canal may be necessary. Putrid admixtures with the fæces are always a serious symptom, and are generally due to the breaking down of malignant tumours or of ulcers, to abscesses, perforations from neighbouring organs, etc.

6. Tenesmus

As a rule, tenesmus indicates an affection of the large intestine. Apart from dysentery, which is almost always accompanied by tenesmus, and from acute intestinal catarrh with copious diarrhœa, the greatest variety of intestinal disorders come into question when tenesmus is complained of. For this symptom may be occasioned by excessive coprostasis, more frequently, however, by proctitis and periproctitis, rectal ulcers or catarrh, hæmorrhoids, prostatitis and prostatic hypertrophy, inflammations and malpositions of the uterus, ovarian tumours, etc., and, finally, by foreign bodies which have entered the rectum from above or below. Especially in children is tenesmus a frequent symptom of acute and chronic intussusception. The condition in each individual case must be determined through inspection and palpation of the rectum and of the rest of the intestinal canal.

7. Gastric Disturbances

It can be readily understood that gastric affections are frequently associated with intestinal disturbances. The converse is also true. As regards acute intestinal affections, especially those accompanied by fever (acute infectious enteritis, perityphlitis, etc.), these require no further explanation. Fæcal vomiting from ileus need only be mentioned in this place, as it will be treated of more in detail in the chapter on Ileus in the special part of this work.

In chronic intestinal diseases, even in those of a malignant nature, the appetite, as well as the other functions of the stomach, may be entirely normal. In intestinal tuberculosis, however, the stomach is often affected. Here the fever which is generally present is an important factor in decreasing the appetite. Frequently, and without special cause, a well-marked chronic gastritis may complicate enteritis (Einhorn, Biedert, Oppler). On the other hand, however, hyperacidity may occur under the same circumstances. Chronic constipation may be associated with glandular gastritis. The periodic vomiting which accompanies intestinal stenoses, especially those of a severe type, is very remarkable. With Nothnagel, we may look upon this as a "regurgitive contraction." (For further details, see chapter on Intestinal Stenoses, special part.) Nausea and vomiting may also occur reflexly, or during long-continued intestinal colics.

Continued bilious vomiting is a very important symptom of deeply seated duodenal stenosis, and will be spoken of at length in the special part of this work.

8. Subjective Abdominal Sensations

Patients sometimes declare their main symptom to be a feeling of pressure and weight in the abdomen. Occasionally they are able to fairly accurately locate the seat of this abnormal sensation. Such subjective symptoms are naturally only of value when combined with the results of the objective examination.

9. Peristaltic Movements

Many patients will state either of their own accord or in reply to direct questions that they occasionally or constantly experience "a crawling, wormlike sensation," sometimes accompanied by severe pain ("peristaltic unrest"). The diagnostic importance of this symptom will be discussed in the section on Inspection.

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CHAPTER IV

THE EXAMINATION OF THE PATIENT

1. Inspection

IN diseases of the intestines the entire body, inclusive of the external anal parts and the rectum should be inspected. Since inspection of the anal region and the rectum is usually associated with palpation of the rectum, it will be considered under that heading.

After we have inquired into the general physical condition and nutrition of the patient, we should inspect his mouth, to ascertain the condition of his teeth, his tongue,* and his pharynx. The patient is thereupon told to disrobe, and is at first examined in a standing position. Any striking variations from the normal should at once be noted (colour of skin, scars, growths, spinal curvatures and protuberances, or depressions of any part of the chest, etc.).

Direct inspection of the abdomen should now begin. The patient should either lie in bed or upon a good elastic couch, with his head extended and the legs and abdominal muscles as fully relaxed as possible. The illumination must be good; daylight is best, but in its stead we can employ gas or electric light, either direct or properly reflected. Like palpation (q. v.), inspection is carried on in two ways: during shallow and during deep respirations.

As regards the *skin* over the abdomen, we have here to look for striæ and venous engorgements. Besides ascites, venous engorgement of the skin results from new growths of the abdomen when these compress the portal system. This condition is not without importance in the diagnosis of abdominal tumours which are deeply seated and palpable only with extreme difficulty. Inspection also enables us to readily detect protrusions or depressions of the abdomen. Protrusions may be localized, or they may extend over the

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* The condition of the tongue in intestinal diseases is of less importance than in gastric diseases. On the other hand, bad condition of the teeth may be a definite etiological factor in gastric or intestinal catarrhs.

entire abdomen; they may result from abnormal accumulation of gas or fluid. We should note small differences in the level of the abdominal surface, especially if some segments of intestines are more prominent than others. Herniæ of the abdomen (umbilical, linea alba, ventral, femoral, and inguinal) are readily recognised, especially when the patient coughs. Usually simple palpation is all that is required to make the diagnosis. The prominence above the general surface of the abdomen of any new growth is very important. On deep inspiration we may convince ourselves of any mobility of these neoplasms, which is a consideration of great value not only for the diagnosis but also for the treatment of the case (operation). It appears important to me, therefore, to point out that *even small new growths are much more readily detected by careful inspection with a good light* (*a very important point*) *than they are through palpation.*

Under especially favourable circumstances (e. g., descensus), and particularly where there is a marked coprostasis, it is possible to recognise isolated segments of the large and small intestine. We find both abnormal abdominal depressions and prominences. Basilar meningitis and lead colic present well-known classical examples of depressed abdomen. Abnormal depressions also occur in marked cachexias, inanition, esophageal and cardial cancers, as well as in other non-stenosing growths in the upper portion of the digestive track. Much interest attaches itself to visible peristaltic movements. These *normally* occur in very emaciated persons, particularly in women who have frequently borne children (*vide* special part of this work), and are then, as Nothnagel[1] correctly points out, limited to the small intestines. Visible peristalsis of the large intestine is therefore a pathological condition. It may be an intestinal neurosis (*tormina ventriculi nervosa*), as illustrated in a striking case which I have recently demonstrated[2], or may constitute an important and, if well developed, a *decisive* symptom of a chronic intestinal stenosis. Since the peristaltic waves are most marked above the point of stenosis, we can in a general way determine the seat of the obstruction. Besides these forms, which really represent but an exaggerated and for the most part a painless type of normal peristaltic movement, there is a second form, the *tetanic intestinal contraction;* this varies according to the emptiness or fulness of the intestines. As instances of tetanic contraction with empty intestines, Nothnagel mentions cerebral meningitis and lead colic. He thinks that in rare cases the contracted coils are visible. This, however, has never been my experience; on the other hand, tetanic contractions with filled intestines are more frequent, and constitute an exceedingly important diagnostic symptom. One may see a rounded elevation suddenly appear in a circumscribed portion of the intestine and accompanied by most severe pain, gradually become more and more prominent, become rigid, and then suddenly sink back again. This is often accompanied by loud gurgling and rumbling sounds and a subsidence of the pain. Nothnagel[1] has very aptly applied the name "intestinal rigidity" to this condition. The gradual increase in intensity, the pause at the acme, and the sudden subsidence may all be better appreciated by placing the hand over the part than by inspection. This form of tetanic contraction always points to an obstruction of the intestinal passage; however, it gives us no information regarding the nature of the obstruction —i. e., whether within or without the intestinal canal, whether caused by a foreign body or by disease of the mucous membrane, etc. The causes of "intestinal rigidity" will be referred to in the special part of the present work.

2. Palpation

A. Palpation of the Abdomen

The great importance as well as the difficulties of palpation have[5] been dwelt upon elsewhere[8]. What has there been stated applies to a greater degree, if possible, to the intestines. In the following the most important points in regard to the technic of palpation are again given.

1. A good couch, not too soft, and accessible from all sides, should be used for the examination (a lounge is preferable to a bed).
2. The patient should lie in the horizontal position, with the head extended and as low as possible.
3. The legs as well as the rest of the body should be as fully relaxed as possible (anæsthetic posture). I have very seldom seen any advantage from the drawing up of the lower extremities which is still often recommended.
4. Palpation should be conducted in a warm room, and only with warm hands, for otherwise the abdominal walls will contract at the slightest touch, and deep exploration become impossible.
5. The attention of the patient is to be distracted from the examination by questioning him regarding his age, heredity, etc., and by having him stretch out his tongue, raise his arms, etc.

6. Palpation at first should include the superficial portions of the abdomen, and only very gradually should the deeper parts be explored.

7. The condition of fulness of the abdominal cavity at the time of examination is very important. The distention of the abdominal cavity with gas or fluid, of the stomach with food, of the large intestine with fæcal masses, and of the bladder with urine, may at times interfere with the examination, but, as will be later shown, this is not always the case. We should therefore make it a rule never to make a diagnosis at the first visit, but should point out the necessity of a further examination after removal of the above-mentioned conditions.

8. Always palpate in the right and left lateral positions, as well as in the dorsal, for frequently growths of the stomach, of the intestines, or of other organs, can not otherwise be palpated. Bimanual palpation is especially to be recommended for the examination in the lateral position.

9. For the better recognition of tumours, enlargements, or alterations in position of abdominal organs, differentiated otherwise with great difficulty, V. Chalapowski, Lennhof, G. Sée, Schuster, Berkhan, and others, have recently strongly recommended palpation in a warm full bath. Although I have had no personal experience with this method, in spite of its inconvenience, it appears to me to possess a number of advantages which would justify a more extensive trial.

In palpating the abdomen we may proceed in one of two ways: Either fix upon and at once explore some point that attracts attention, or examine systematically. The former method is to be recommended only to the experienced, while the latter is to be recommended to the beginner. I would advise the following method of procedure: Examination of the abdominal wall for œdema, emphysema of the skin, excessive fat, lipomata, etc.; examination of the epigastrium, paying special attention to splashing and succussion sounds, etc.; examination of the right and left lobes of the liver, the region of the small intestines from the pylorus downward to the umbilical region. The hernial canals should be palpated; also the region of the cæcum (McBurney's point midway between the anterior superior spine and the umbilicus). From the cæcum the examination is to be continued along the ascending, transverse, and descending colon, and the sigmoid flexure. The ascending and descending colon are best palpated in the right and left lateral postures respectively. At the same time the condition of the kidneys (dislocation, fluctuation, and tumours) and of the spleen may be determined.

This plan is of course, to a certain extent, schematic, and assumes that the intestines are in their normal position. We must, however, reckon upon possible anomalies of single segments, especially those of the very mobile portions of the large intestines. (Compare further remarks upon this on page 21 et seq., and also the chapter on Displacements of the Intestines, in the special part of this work.)

Under favourable conditions it is undoubtedly possible to palpate certain segments of the intestines. Obrastzow[4], who has thoroughly studied the technic of this subject, has obtained remarkable results by palpation. He was able to locate the cæcum in 51.47 per cent of the men and in 58 per cent of the women he had examined; the transverse colon in 23 per cent and the sigmoid flexure in 65 per cent of all his cases. Unfortunately these results are not accompanied by post-mortem records, and therefore they lose much of their statistical value. As pointed out in the oft-quoted instructive treatise of Curschmann, the greatest caution must be exercised in accepting palpatory results of the large intestines, since the latter are so frequently displaced. Nevertheless, we may safely assert that the sigmoid flexure is the most easily palpated segment, the transverse colon the most difficult, while the cæcum occupies a position between the two. It is frequently possible to palpate the sigmoid flexure and the ascending colon from their being filled with scybalæ; upon palpating the lower portion of the ileum or cæcum, the impression is conveyed as if of a thin pasty mass under the fingers, and one can hear and feel the gurgling sounds, formerly considered an important symptom of typhoid fever. These signs, however, are to be accepted in a diagnostic sense only after most careful consideration. Abnormal thinness of the abdominal walls and distention of the bowel by fæces render palpation of the large intestine much easier. In the presence of such conditions, it is, I believe, possible to map out the entire large intestine. To determine intestinal displacements it might even be advisable to bring about an artificial coprostasis.

Recently, Edebohls[5] has claimed that the vermiform appendix, especially in women, is often palpable, and that from the condition found one can tell whether in a given case the appendix is a normal or an abnormal one. He recommends that one should press as deeply as possible toward the posterior wall of the abdomen and floor of the pelvis, keeping to the outer side of the iliac artery. I have never succeeded in satisfactorily palpating the appendix. In my experience, it is easier to palpate isolated coils of the small intestines, particularly in enteroptosis.

This, however, is of no practical advantage, for it will rarely be possible to recognise what particular segment is felt. I would here remark that Obrastzow[6] points out the importance of palpation of the ileum for the diagnosis of typhoid, claiming that the gut appears thickened, uneven, and painful.

As in the stomach, so also in special palpation of isolated intestinal segments the following points come up for consideration.

(*a*) *Sensitiveness to Pressure; Pain* (*Circumscribed or Diffuse*)

We shall first give a few practical preliminary remarks. All portions of the intestines are painful upon rough handling, the cæcal region more so than the rest; furthermore, owing to the adjacent sympathetic fibres, pressure over the large blood-vessels causes pain, especially in women whose abdominal walls are relaxed from frequent childbirths, and in very emaciated men. In the palpation of isolated intestinal areas of anxious and excited individuals I have frequently met with a "pseudo-painfulness." It is well to have the painful areas pointed out and to re-examine them after a few moments. We can thereby convince the patients themselves of their error. In this connection I wish also to call attention to a general hyperalgesia of the abdominal wall not infrequently met with in neurasthenics.

Practically, we should distinguish between pressure sensitiveness and pain. Thus, an acutely inflamed appendix, or, in acute dysentery, the sigmoid flexure and the descending colon are painful, whereas in chronic enteritis the large intestine is but slightly sensitive to pressure. The differentiation of such degrees of sensitiveness must be carefully practised and studied. Very important, too, is the differentiation between a localized and a diffuse pressure sensitiveness. In ulcer of the duodenum we meet with a very circumscribed area of pain, beyond whose limits pressure made with the necessary precaution is absolutely painless; on the other hand, in appendicitis with diffused peri-appendicular abscess the entire region of the cæcum, or even the entire ascending colon, may be painful on pressure. Again, in general peritonitis the entire abdomen is painful even to the slightest touch. Multiple circumscribed sensitiveness is found in catarrh of the small intestines complicated by follicular ulcers (for the most part tubercular); it may, however, be absent. When present, it can, when viewed with a certain amount of reserve and in connection with other symptoms, acquire a diagnostic value. I have often observed a diffuse pressure sensitiveness in chronic sigmoiditis. This I regard as an important symptom to which little attention has been paid. I will refer to it more in detail in the chapter on Enteritis (special part). A pressure sensitiveness, at first localized, but later diffused, may coexist with malignant growths, stenosis from other causes, intussusception, and volvulus. Further details of this subject must also be reserved for the special part of this work.

(*b*) *Splashing Sounds* (*Clapotage*) *; Succussion Sounds*

In my experience, intestinal splashing sounds occur only in the large intestine, and only under certain conditions. As far as I can learn from the literature, this subject has received but little attention; it therefore seems proper to consider it more in detail. A certain relaxation of the intestinal walls as well as the presence of fluid or thin pasty contents are necessary for the production of splashing sounds. Furthermore, the abdominal walls must be sufficiently yielding, so that the wave of contact may be readily transmitted to the intestines. Thinness of the abdominal wall and laxity of the intestines are normally present in women who have borne many children. It is different, however, with regard to the physical state of the intestinal contents. From the investigations of Macfadyen, Nencki, and Siebers (p. 45) we know that normally the contents of the lower part of the ileum are of a thin pasty consistency; as such they enter the cæcum, and there gradually assume a firm cylindrical shape. *Normally*, therefore, a splashing sound is never elicited beyond the cæcum, or the ascending colon.

To make certain whether or not substances of pasty consistency can cause splashing sounds in the large intestines, I directed my former assistant, Dr. Ehrlich, to ascertain whether splashing sounds could be elicited in the large intestine after injections of large quantities of thick pasty soups. It was found that these sounds could be distinctly elicited in the sigmoid flexure, and in two cases in the cæcum, and more particularly in the transverse colon. Marked liquefaction of the contents of the large intestines is still more favourable for the production of splashing sounds; under such circumstances they may be heard even over extensive areas.

It appears possible, therefore, without special preparation, to determine *approximately*, the position of one or more segments of the intestines by means of the splashing sounds. Naturally, this can only be accomplished with normal situation and an absence of gastric succussion sounds.

I was the first to recommend methodical filling of the large intestine with measured amounts of water for determination of the splashing sounds[7]. This method is somewhat cumbersome, and as yet not well known; it does, however, give us some diagnostic data. If after thoroughly emptying the intestines of a healthy individual, we allow lukewarm water to flow *slowly* into the rectum through a soft-rubber tube and funnel, all segments of the large intestine will gradually fill up. After 500 to 600 cubic centimetres have been introduced, a splashing sound, more or less distinct, may be obtained, at first in the region of the sigmoid flexure, but later also in the transverse colon, and finally in the cæcal region. Under favourable conditions a slight succussion sound can be heard upon changing the position of the body. In marked atony of the large intestine we will obtain a splashing sound in the above-mentioned places after only 200 to 300 cubic centimetres have been introduced. Should, however, the splashing sound be heard at a point other than where it normally ought to be (e. g., far below the umbilicus), it would indicate displacement of the segments of the large intestine in question. As in the stomach, downward displacement or a dislocation of the segments of the intestine frequently coexists with atony. J. Friedenwald[8] has also employed this method and confirms its diagnostic value.

Just as we can obtain splashing sounds in the intestines filled with water by striking them with the tips of the fingers, so we may also readily elicit succussion sounds wherever there is an excessive relaxation or a dilatation of the intestinal walls by shaking the patients or by having them rapidly change their position. Succussion sounds are very readily heard in the sigmoid flexure or the descending colon.

(c) *New Growths; Fæcal Tumours; Adhesions*

New growths occur in all parts of the intestinal canal, but increase in frequency as we go downward. Malignant tumours (cancer, sarcoma, lymphosarcoma, tubercular tumours) are met with more often than benign tumours (myomata, fibromata, polyps, adenomata, angiomata, syphilomata, etc.). If in the following discussion we speak of tumours in general, we nevertheless have the malignant new growths principally in view.

When a new growth is found in the abdominal cavity and question of its connection with the intestines is raised, we should first of all consider all those other organs to which the tumour might possibly belong. Palpation alone is generally not sufficient; other methods of examination—the examination of the stomach contents, inflation of the stomach and intestines, injection of water, as well as other clinical phenomena (urine, blood, and especially examination of the genitals)—must be brought to our aid. As in other abdominal tumours, in intestinal tumours *the position*, *mobility*, *size*, *consistency*, *sensibility*, *and respiratory mobility*, have to be determined.

As regards the position of a new growth, that is determined in the first place by the normal position of the intestinal segment with which it is connected. The position will depend very much upon the mobility of the intestinal segment in question. For instance, a cancer of the pylorus may be found in the region of the cæcum, or a perityphlitis below the right lobe of the liver.

To begin with the small intestines, the duodenum is comparatively firmly fixed, so that its tumours can very seldom cause marked alterations in its position.

Owing to their large and more freely movable mesentery, tumours of the jejunum and of the ileum have a somewhat greater mobility. As Nothnagel[9] very correctly observes, this may lead to great difficulty in the differentiation between tumours of the large and small intestines. The cæcum and the ascending and descending colon are the least movable segments of the large intestine, while the transverse colon and the sigmoid flexure have the widest range of motion. In tumours of these segments there will very probably be a dislocation of the parts. The tumour itself may tug upon the portion of the intestine, dragging it laterally or downward; or else the bowel may become distended above the site of the stenosis, and becoming abnormally loaded, may drag the intestinal segment at first partially, later completely downward; finally, in consequence of beginning emaciation, there may be a relaxation of the fixation bands with a resulting alteration in position of the intestinal segments.

Furthermore, we must distinguish between passive and active (manual) mobility. I have found passive mobility of intestinal tumours to be rare; active motion occurs either not at all, or else to a very striking extent. The mobility of the tumour will depend principally upon whether a ptosis of the bowels has taken place, and, further, upon the presence or absence of adhesions. The nature of the tumour itself is also very important. Regarding the *size* of the tumour, this, as in the stomach, will vary considerably, according to the stage of the process. I have seldom observed intestinal growths acquire such dimensions as those of the stomach; usually they are very much smaller. Sarcomata and benign growths form an exception; they may reach enormous dimensions.

The consistency of the growth depends upon its origin and malignancy. Cancers are hard and nodular; sarcomata hard and smooth, and they not infrequently have a central soft or even fluctuating area. Benign growths, as well as intussuscepted portions of the intestines, are smooth and uniform, while abscesses and cysts always impart the feeling of fluctuation to the examining finger.

The sensitiveness of intestinal growths is likewise subject to the greatest variations. They are, perhaps, never entirely painless; the pressure sensitiveness of benign growths is very much less than that of malignant ones. Upon the whole, however, no great weight need be attached to the degree of sensibility of the intestines.

All parts of the intestines are more or less movable with respiration, most so where the intestinal wall is thin, and poor in fat; on the other hand, adhesions of organs directly connected with the diaphragm (liver, stomach, and spleen) may have a disturbing influence. Very little diagnostic significance attaches itself to the respiratory mobility of intestinal growths. The question of existing adhesions is best determined under narcosis.

Fæcal tumours play such an important part among intestinal growths, and so frequently give rise to errors and confusion in intestinal diseases, that they require special consideration. They may give rise to errors in two ways: first, in simulating new growths, and, secondly, in causing existing new growths to appear larger than they really are. In such cases, usually, the diagnosis can not at once be made; we should allow ourselves more time and observe the effect of internal and external laxative measures. As a rule, the consistency of the growth in question will give us a useful diagnostic hint. Apart from the so-called enteroliths, fæcal tumours are generally of a somewhat doughy consistency, and retain the impression of the finger, especially after rectal enemata of oil or of soap (which tend to soften them). Even after such enemata the peripheral portion of the fæces may remain hard (*Randkoth*) and simulate a tumour, notwithstanding that frequent voluminous evacuations (*Centralkoth*) have taken place. This experience, which has been that of absolutely reliable clinicians, calls for the greatest caution in the determination of the nature of doubtful tumours. The situation can usually be determined, especially upon repeated examinations, although a very instructive case described by Nothnagel shows that even then errors can not always be excluded. The history, the status, the clinical course, in short, everything in Nothnagel's case, indicated cancer of the cæcum, while the autopsy showed an ulcerative tubercular stricture in the beginning of the ascending colon.

Gersuny [10] believes that the difficulties in diagnosis so often caused by fæcal tumours have been overcome by the discovery of a peculiar symptom which he describes as "the adhesive sign" (Klebesymptom); viz., if the finger be very firmly pressed upon the fæcal mass, the intestinal mucous membrane will become adherent to the viscous fæces, becoming free again when the pressure is discontinued. Gersuny states that we can feel the mucous membrane loosening itself, and that such a sensation is characteristic of a fæcal tumour. Hofmokl [11] could not, however, convince himself of the existence of this "adhesive sign," and, like myself, regards the impressibility of the fæcal tumour as the most characteristic symptom.

Under favourable conditions adhesions between the portions of the intestines may be palpated and recognised as cicatricial strands; more frequently, however, they are suspected rather than recognised.

B. Inspection and Palpation of the Region of the Anus and Rectum; Examination with Rectal Bougies

Palpation is the most certain method of acquainting ourselves with diseased conditions of the rectum; in many cases, however, it must be supplemented by inspection and the use of bougies. Palpation of the rectum should be preceded by inspection of the anus. The patient is to be examined either in the lateral or in the knee-chest position. Personally, for complete examination of the rectum and anus I prefer the latter position. With good illumination we can inspect external hæmorrhoids, fissures, intertrigo, pruritus ani, furuncles, phlegmons, external fistulæ, etc. At the same time we can at once learn whether or not any pathological secretions (blood, pus, or mucus) come from the anus. Inspection is best followed immediately by palpation. When a patient complains of rectal trouble, rectal palpation should never be neglected. It not infrequently happens in very timid or prudish persons, especially women, that we meet with opposition, and we should then, quietly but firmly, explain the absolute impossibility of forming an opinion without direct local examination. Even in apparently harmless cases I do not hesitate to point out the possible existence of serious disease of the rectum. This very quickly has the desired effect.* Palpation of the rectum is also absolutely unavoidable in other *chronic* stomach and intestinal affections, which at first glance may appear to have nothing in common with the rectum.

In my Diagnosis and Therapeutics of the Diseases of the Stomach (Part I, p. 72) I have emphatically stated that we ought never to be satisfied with the mere diagnosis "hæmorrhoids," but should always explore the rectum very carefully. *Sometimes a human life may be lost through such utterly inexcusable negligence.*

Technic of Rectal Palpation

As a rule, the examination is conducted in the knee-chest or lateral position (see above).

The finger should be well anointed with borovaselin (oil or glycerin are less commendable).† The examination itself must never be a forcible one, and should consist in gentle rotatory movements, and where the patient already complains of rectal pain should be carried out with particular care. In some cases cocainization of the rectum may be advisable, though I find it can generally be dispensed with.

Passing the examining finger slowly forward, we learn the condition of the mucous membrane, the presence of foreign bodies or of new growths, and the patency of the rectal lumen; furthermore, the condition of the prostate, and in women, such anomalies of the sexual organs as can be recognised by palpation through the rectum.

If the disease process be situated high up, and is entirely inaccessible, or accessible only with great difficulty in either of the two mentioned positions, we may reach it with the patient in the dorsal position and by suitable pressure in the left iliac region. In like manner we may succeed with the patient in a standing position, when the examiner should be on one knee and support the elbow of the examining hand upon the other knee bent at a right angle. At the same time the patient should be asked to make strong pressure downward.

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* Other circumstances may at times lead to difficulty in the exploration of the rectum. Kelsey, the experienced New York rectal specialist, relates the following characteristic story: "A foreigner told me, when I proposed it [rectal examination], that he had entirely too great respect for me to allow such a thing. My only answer was that I had too great respect for myself to treat him without knowing what was the matter. That helped, and," Kelsey concludes," we parted amicably."

[† To prevent the fæcal odour from clinging to the finger after a rectal examination, it is a very effectual plan before the "preliminary anointing" to liberally scrape ordinary soap under the free edge of the finger nail and between the cuticle of the finger and the matrix of the nail. The so-called "finger cots" protect the examining finger from infection and fæcal smell, and when made of the proper thinness do not interfere with the palpatory sense.—Tr.]

At the time of the first examination it is very desirable to make the digital exploration of the rectum as carefully and fully as possible, so that we ascertain all that can be learned through palpation.

In some cases the introduction of one finger is not sufficient to palpate the entire diseased area. For this class of cases Simon, many years ago, recommended the introduction of the entire hand into the rectum. This procedure, which is by no means harmless, can of course be carried out only under general anæsthesia. In consequence of the resulting relaxation of the sphincter, one or two fingers usually suffice for the exploration, and the entire hand is necessary in isolated cases only. In rare cases, however, bloodless distention, or even slitting up of the sphincter (sphincterotomia posterior recti), must be undertaken. Since both of these are usually preparatory to operations on the rectum, we must refer the reader for further details to surgical text-books.

Examination under narcosis is also indicated, either where the nature of the disease can not be otherwise determined because of excessive painfulness of the exploration, or else where the disease, being known (e. g., cancer), the determination of its extent, particularly upward, is attended by great difficulties, or, finally, where new growths are barely accessible to the finger.

If inspection or examination with bougies is also required, it is best to proceed with these immediately after palpation. The patient is thus spared the excitement attendant on every rectal examination, and, what is most important also, the uncertainty of diagnosis.

Ocular inspection of the rectum is best conducted on an operating table, in the lithotomy position, whereby the sacrum lies on a level with the examiner's chest. The knee-chest position may also be used. The examination should be made with the aid of a suitable speculum. The number of these specula almost equals those for the vagina. They are all best described in the catalogues of instrument makers. For practical purposes most of them are too complicated and clumsy. With the observance of the precautions described below, one usually succeeds with a simple Sims's, Simon's, or a Czerny's speculum, the latter having given me excellent service on account of the shortness of the one end. A bivalve speculum is also very good. With proper illumination, one obtains a satisfactory picture of the internal rectum.

Palpation should always precede the introduction of the speculum. The presence or absence of fæces in the rectum can thus be determined. If present, they must be removed through irrigation with lukewarm water or weak lysol solutions, or else manually.

The introduction of a speculum is very much facilitated by the relaxation of the rectum. This is readily accomplished by the knee-chest position, in which the abdominal organs fall forward and negative pressure in the rectum results; the latter fills with air and its walls stand apart. I have convinced myself that deep inspiration combined with passing the finger into the rectum very much facilitates the passing of the speculum. If the instrument be uniformly and liberally anointed with a fatty substance, its introduction will be attended by no difficulty. With a good natural or artificial light one can carefully inspect all pathological conditions of the rectum. This has recently led Kelly[12] to recommend the introduction of cylindrical specula with a uniform diameter of 22 millimetres and a length varying from 14 to 35 centimetres, and furnished with obturators. They are introduced with the patient in the knee-chest position. As soon as the speculum has passed the sphincter the rectum fills with air, and an entirely unobstructed view of the rectal cavity is thus obtained.

Attempts have been made to illuminate the rectum just as in other cavities in the body. J. Leiter, of Vienna, constructed an instrument for this purpose[13], which, however, appears to have been put to little practical use. After introducing the speculum into the rectum, one may employ either reflected electric illumination, or more simply, direct electric illumination.

Herzstein has lately constructed an instrument which I have found very useful for the examination of the rectum (Fig. 11). It consists of several metal tubes (*A*), varying in length from 10 to 34 centimetres, and furnished with metal obturators (*B*) for the purpose of easier introduction. The screw (*d*) fastens the obturator to the metal tube. The illumination is supplied by the well-known Caspar electroscope (*C*). The longest tubes enable one to reach the sigmoid flexure. The field of vision is kept clear of mucous fragments or fæces by cotton applicators 20 and 35 centimetres long. To facilitate the introduction of this instrument I have had the obturators made conical, and in order to determine which portion of the rectum the speculum has reached I have had a centimetre scale marked upon the tubes.

Inspection of the rectum, in the first place, enables one to detect acute and chronic inflammations of the mucous membrane. In acute cases the mucous membrane has not the normal pale appearance, but appears swollen and strongly injected; in chronic inflammations it appears relaxed, swollen, and velvety, is covered with firm mucus, and easily bleeds. With good illumination the follicular swellings become distinctly visible. In well-pronounced cases, a muco-purulent secretion trickles over the surface. As a rule, these inflammations are not primary, but occur in connection with other rectal diseases. Furthermore, on inspection one can distinctly see ulcerations (fæcal, dysenteric, tubercular, gonorrhœal, hemorrhoidal, syphilitic, etc.), and, in children, follicular ulcers. When typically developed, these different forms can be distinguished from one another. Rectal fistulæ (internal and external), hemorrhoidal veins, polyps and other growths, strictures, etc., can also be seen. Since these various affections are treated of at length in the second portion of this work, we may here content ourselves with this brief reference to the subject.

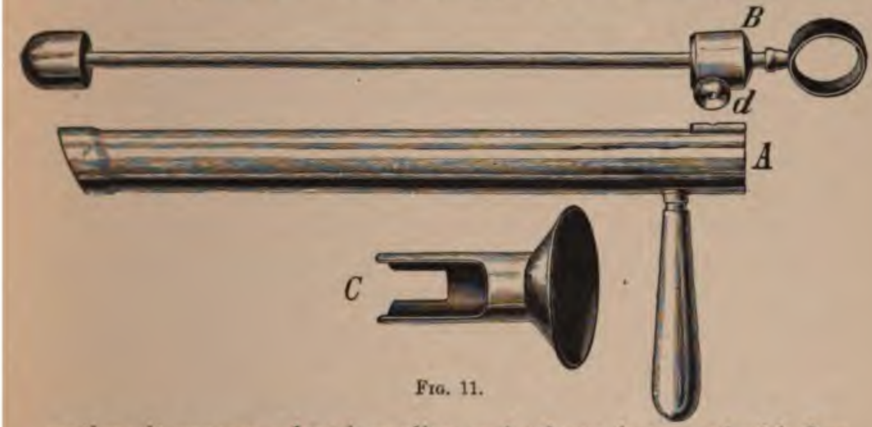

FIG. 11.

By bougieing of the rectum and of the parts above, we may sometimes learn whether or not that gut and the sigmoid flexure are patent. For this purpose we employ either soft-rubber bougies,* or else the so-called English [known here in the United States as the French sounds] or hard-rubber bougies, or, finally, though more

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* Those with a spiral obturator, as devised by Hahn, are particularly good, because they combine great elasticity with firmness.

rarely, the so-called Trousseau's sounds, with olive-shaped ivory points that can be screwed on and off. The softer the instrument employed for diagnostic purposes the less the danger. When sounding the rectum for the first time, I use only soft-rubber sounds or bougies. The question how far a bougie may be introduced is very important. Some authorities state that it can not be passed beyond the sigmoid flexure. According to my own investigations this is incorrect, for by abdominal palpation of sounds which I had introduced I have been convinced that, if the colon be previously distended by water or air, even soft Nélaton sounds *can* be passed into the descending colon. I purposely employ the word *can*, for our success depends principally upon the sound passing through Nélaton's sphincter into the sigmoid flexure. In some cases this is accomplished very readily, in others only by the aid of one or two fingers in the rectum acting as a guide to the instrument. Should this last precaution be neglected, flexible instruments curl up in the wide ampulla or impinge against its walls, thus making forward passage impossible. On account of the acute bend which the splenic flexure makes at the junction of both segments of the colon, passage into the transverse colon, on the contrary, is impossible.

Bougies and sounds should be thoroughly oiled before introduction; the latter procedure must be attended by the greatest precaution. All force must be carefully avoided. This rule applies particularly to all sounds which are not absolutely soft, for during simple sounding of the normal rectum perforations have occurred.

The *diagnostic* value of rectal bougieing is not great. We can not learn more from bougieing, at least of the rectum, than we can from digital and ocular examinations. On the other hand, only through sounding can constrictions of the sigmoid flexure or of the descending colon be diagnosed with certainty. Such examinations, however, require the greatest possible care, and I would again recall the advantages of the above-mentioned rubber bougies.* *A stricture can be diagnosticated with certainty only when, upon repeated examinations, the passage of a sound imparts the impression of its*

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* Kuhn, of Giessen, recently devised metallic spiral sounds for the sounding of the stomach, pylorus, duodenum, and the large intestine. From these sounds he hopes to learn much of value for the diagnosis and therapeutics of the said organs. Since no evidences of the merits of this method have as yet been advanced, we will not discuss it, nor the method of Hemmeter for the intubation of the duodenum.

always having to overcome a resistance at the same point, and when, after the suspicious point is passed, the withdrawal of the sound also gives one the impression of passage through a narrow portion.

Aside from diagnostic purposes, rectal bougies are also employed for the dilatation of strictures. This will be referred to in the proper place in the second portion of this work.

3. Percussion

In general, percussion gives us less tangible results than palpation. Nevertheless it is of value as completing and controlling the latter procedure, and it should therefore never be omitted.

Percussion of the intestines should always be finger percussion, never hammer percussion, for with the latter finer distinctions are very easily overlooked. We should always percuss lightly, for then only, if at all, can differences in tone be clearly brought out. In the empty state of the intestines percussion gives the best results. It is sometimes of importance to compare the result of percussion of the empty with that of the full intestines (and stomach). Normally, as in the stomach, the percussion note is tympanitic, but varies in pitch in the different segments. On account of the descending colon being filled with solid matter, palpatory percussion, according to Leo[14], almost constantly gives a dull tympanitic tone in the left hypochondrium as compared to that obtained on the right side. The diagnostic value of percussion is limited to those cases in which we find distinct variations from the normal tone. This occurs with free or encapsulated fluids in the abdominal cavity, and also in excessive meteorism. The results from percussion in ascites are so well known that detailed discussion is unnecessary. We wish, however, to speak of the differentiation of air in the abdominal cavity from that in the intestines. Whereas in the former instance the percussion note has everywhere a uniformly tympanitic sound, and hepatic and splenic dulness disappear, in intestinal meteorism the percussion note varies from place to place and from time to time.

The percussion note over large tumours of the intestines (including fæcal tumours) is flat, provided they are not covered by intestinal coils that contain air. In the presence of moderate amounts of fluid in the abdomen, tumour percussion in various postures of the patient may also yield useful results. If the abdomen be tapped before percussion the results will naturally be more satisfactory.

By filling the intestines with air or water we can establish conditions favourable to percussion (see pages 74 and 85).

In ileus, in order to distinguish between widely distended intestinal coils and those of smaller calibre, a few authors, particularly Curschmann[15], have recommended pleximeter percussion.

4. Auscultation

Of all physical methods of examination, auscultation has the least diagnostic value. It is true that sounds (borborygmi, gurgling, etc.) are heard in the most varied diseases of the intestines, but the nature of their production is so atypical that little can be learned from them. In stenosis of the large intestines we meet with intestinal sounds (especially frequently recurring intestinal sounds) on a most extensive scale, but compared with the data obtained from palpation, or even inspection, their diagnostic value is minimal. On the other hand, the absence of all intestinal sounds, together with total absence of all intestinal movements, is of diagnostic value in perforative peritonitis (E. Wagner[16]).

The well-known ileo-cæcal gurgling of typhoid fever to-day possesses an historical value only. All experienced physicians agree that it has no practical value. Regarding succussion and splashing sounds, compare above (page 73).

5. Inflation of the Intestines

Inflation of the intestines can be accomplished either through the introduction of mixtures which form carbonic-acid gas (bicarbonate of soda and tartaric acid, von Ziemssen[17]), or of carbonic-acid gas from fluids in retainers (O. Rosenbach[18]), or else from inverted siphons of carbonic-acid gas[19], or finally through the introduction of atmospheric air by means of a double balloon bulb, such as is used with a spray apparatus (Runeberg[20]). Of all these methods the last mentioned is certainly the simplest and best; we can inflate the intestines with as much or as little air as we wish. Should there be any occasion for measuring the amount of air introduced, we may employ a syringe of known volume and introduce the air from this (Damsch[21]).

How far can the air be introduced? Most authors, particularly von Ziemssen and O. Rosenbach, claim that the air can not pass beyond the ileo-cæcal valve; whereas Damsch has shown that with slow inflation and the employment of more than one metre of air, the valve may be overcome and even the small intestines inflated. In view of similar successful attempts made with large quantities of water (von Genersich[22]) we must at once admit the possibility of overcoming the resistance of the valve with air. Both methods are very heroic, and have, besides, no advantage over the introduction of small quantities of air or water.

Upon the whole, therefore, we shall have to remember that, as ordinarily practised, the introduction of air distends the large intestine only.

The technic of rectal inflation is very simple. For this purpose a soft stomach or rectal tube, or, in case of necessity, even the rubber tube of an irrigator, may be used. A well functionating double balloon bulb is attached to the tube. Before introducing the rubber tube we must see whether there is much fæcal matter in the rectum; if so, it must be removed. Small quantities of fæces need not be taken into account. To learn if air passes into the intestines, we should auscultate in the left iliac region; a hissing sound made by the entering air can here be distinctly heard. Practically it is very important not to inflate the intestines too greatly, for finer shades of difference in sound are best appreciated with slight inflation. It is often useful to combine inflation of the stomach with that of the intestine.

Inflation of the intestines has a threefold practical diagnostic signification; it enables us to diagnose stenoses with a certain amount of probability, sometimes even with certainty; furthermore, it makes clear displacements of the intestines, especially of the movable portions; finally, it is of value in locating tumours and in determining their mobility.

Regarding the diagnosis of stenoses, we may remark that normally, in compliance with physical laws, the entire large intestine forms a uniform prominence; in stenosis of a segment the infra-stenotic portion only can be inflated, and will therefore appear more prominent, while the supra-stenotic portion will be very little or not at all distended. Furthermore, if the inflation be continued, the air will return and pass out *per anum* alongside of the tube, and severe pain will be apt to follow. The results obtained from percussion in intestinal stenoses are also important; while we still have the usual tympanitic sound above the stenosis, we will get a deeper and more sonorous one below it, and after all the air has passed out both sounds will again be uniform. When the inflated air presses upon the stenosed parts it is sometimes possible to hear a peculiar long-drawn sound that may also be of diagnostic value.

Great as may be the value of this method, for the development of which much credit is due to von Ziemssen, it should nevertheless be remarked that reliable results are obtained therefrom only in very marked stenoses. In these cases, however, the other symptoms of intestinal obstruction are so well characterized, that, after all, intestinal inflation should be employed mainly as a confirmatory method. For obvious reasons it frequently gives negative results in the beginning of a stenosis.

A further advantage of the method lies in the possibility of diagnosticating, with more or less certainty, changes in the position of various segments of the large intestine. If, for example, after moderate distention of the large intestine a transverse protrusion is seen below the umbilicus, or even immediately above the symphysis, and the same result obtains after repeated examinations, we are safe in assuming that a sinking of the transverse colon (*coloptosis*) has taken place. The sigmoid flexure sometimes becomes enlarged or dislocated, or both, so that the greatest difficulty may be experienced in differentiating it from the transverse colon.

Finally, as is well known, in inflation of the intestines we have a means of ascertaining with certainty the position of questionable intestinal tumours, and of distinguishing between these and tumours of other organs (e. g. the kidney), since the latter disappear gradually with inflation, whereas the former (tumours of the intestine) remain as perceptible as before. Nevertheless, this symptom can only be taken into account where a tumour previously perceptible completely disappears. In like manner intestinal tumours may frequently be very clearly differentiated from those of the liver.

6. The Injection of Water per Anum

The injection of water *per anum* is principally employed to locate an intestinal stenosis (Simon), or to establish the relation of a new growth to surrounding organs (Minkowski[23]). In the latter case it is well to combine the procedure with inflation of the stomach.

Here, according to Minkowski, the abdominal tumour slips back into the position normally occupied by the organ to which it belongs. Here, also, it is frequently well to combine the distention of the stomach by air or water with similar distention of the large intestine. The recognition by means of distention of a stenosis—especially one occurring far down—is based upon the fact that under normal conditions 2 to 5 (!) litres can be passed into the intestines, whereas in deep stenosis very much less can be introduced. This method is unreliable if for no other reason than because the toleration of the rectum for quantities of water varies considerably (at least according to my experience). The water is introduced by means of an irrigator armed with a calibrated tube or else by means of a Hegar's funnel.

7. Test Lavage of the Intestines[24]

By this I mean a uniform irrigation of the mucous membrane of the entire large intestine with water, for the purpose of recognising anomalies of the same (mucus, pus, blood, or even fragments of tumours) in the returning water.

Technic.—The following rules are given for *test lavage* of the intestine. It is best carried out with the patient in the lateral position, the intestines being previously emptied. I use a soft-rubber tube, not too small in calibre, and from about 70 to 80 centimetres in length, armed, as in lavage of the stomach, with rubber tubing and a large funnel. The tube is well greased, and after its introduction into the anus is gradually pushed forward, while the funnel, filled with lukewarm water, is at the same time slowly raised. In suitable cases—as can be proved by palpation through the abdominal wall—we are sometimes able to introduce the tube into the sigmoid flexure, or even beyond. As soon as marked tenesmus occurs (not infrequently after more than a half litre has been introduced) the funnel should be lowered, and the water thus siphoned off should be carefully inspected and preserved in clean vessels for macroscopic and microscopic examination. Normally, the water returns clear, or only slightly cloudy from admixture of very minute particles of mucus, epithelium, or small particles of fæces. It is very different, for example, in catarrh of the large intestine. Here the wash water to a greater or less extent contains large and small shreds of mucus. Their quantity serves as a good criterion of the progress and the extent of the disease. We have no simple or easier method than this for diagnosticating membranous enteritis. This procedure, which I have used for a long time, has also given me excellent results in detecting suppurative and ulcerative processes accompanied by hæmorrhage. I have often found pieces of exfoliated mucous membrane in the wash water, and though I can not speak from personal experience, it appears possible to readily obtain in this manner fragments of tumours for examination. Where it is desirable to make a microscopical examination, this procedure possesses the advantage of absolute cleanliness, a fact which is very advantageous considering the offensive smell of fæces. Besides, the isolation of pathological substances (free from fæcal matter) makes the microscopical examination much easier.

8. Electric Trans-illumination of the Large Intestines

Heryng and Reichmann[25] were the first to employ diaphanoscopy for the determination of the limits of the large intestine. They used a black elastic intestinal bougie, to the end of which a small Edison lamp was fastened. Before introducing the bougie, 1,500 centimetres of lukewarm water were poured into the rectum. By using a lamp of 25 volt ampères (equal to eight standard candle power), the contour and course of the transverse colon were distinctly mapped out. With the development of the method, Heryng and Reichmann hoped it would be possible to approximately determine doubtful tumours of the abdominal cavity, but thus far this expectation has not been realized.

APPENDIX

The Employment of Röntgen Rays in the Diagnosis of Intestinal Diseases

Hitherto the results in general from skiagraphy in the diagnosis of abdominal diseases have not been very promising; especially has it been impossible to positively diagnosticate abdominal tumours that could not also be diagnosed by the usual methods. On the other hand, the location of metallic foreign bodies in the large intestines by means of the Röntgen rays appears to me to be very plausible. I am convinced that we can see certain portions of the large intestine—e. g., the descending colon—and especially distinctly recognise the haustra coli. It is therefore not beyond the limits of possibility that we should be able to render displacements of the intestines visible to the eye. It remains to be seen how much more favourable conditions will be created through the introduction of metallic sounds and the like into the rectum. Since the kidney (particularly the left) and the spleen can frequently be distinctly recognised by the fluoroscope, it might be possible, under favourable conditions, to make the differential diagnosis between tumours of the kidney and of the intestine, or of the liver and the spleen.

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CHAPTER V

EXAMINATION OF THE FÆCES

Preliminary Remarks.—The examination of the fæces is an integral factor in the diagnosis of intestinal diseases. We learn from it pathological changes in the secretive, absorptive, and motor functions of the stomach and intestines, and ascertain the foreign substances that are mingled with the intestinal contents. Frequently also the nature and location of intestinal diseases can thus only be correctly appreciated and diagnosticated. The diagnosis of entozoa must be made almost entirely from the microscopical examinations of the dejections.

In spite of all this, we are safe in stating that even at the present day clinical examination of the fæces is not universally practised. The cause for this is very clear. In the first place, the repulsive nature of the material itself, so disagreeable to the olfactory nerves; then, again, the unpleasant manipulations that the transport and examination of the material entail upon the layman and the physician; and last, but not least, the fact that fæcal examinations rarely yield decisive diagnostic results. The first two objections mentioned are undoubtedly justifiable; the latter objection is also valid to a certain degree. But the principle, *salus ægroti prima lex esto*, should help us to overcome all hesitancy and difficulties. As regards the diagnostic value of fæcal examinations, it must be admitted that a single examination is only rarely sufficient to make clear the nature of the disease. Still, this applies equally well to all other secretions and excretions—e. g., sputum, stomach contents, urine, and vaginal secretions.

In clinics and dispensaries the facilities for macroscopical and microscopical examination of the fæces are usually very easily arranged. In private practice, on the contrary, it is usually best to make the first gross examination in the dwelling of the patient. Should we find anything unusual, or should we for any other reason wish to make a microscopical examination, we can select the parts which appear pathological, and preserve them in a small wide-mouthed bottle with a glass stopper. Only where exact chemical analysis (perhaps for nitrogen or fat) is required, will large quantities of fæces be necessary. In some cases—for instance where we suspect amœbæ—it is very desirable to examine the fæces fresh and at the body temperature, if possible. This is readily accomplished by warming the bedpan and bringing the material into a bottle, also warmed, and wrapped for safety's sake in cotton. If we have an incubator we can place the material in it until we are ready to make the examination, otherwise some artificial means for maintaining the warmth of the fæces can easily be devised. In some cases it is interesting to follow the development of gases from thin stools placed in a warm atmosphere, and to note the influence exerted by certain internal medication upon such gaseous formation. For this purpose we may use the so-called "fermentation tubes" of Einhorn and Fiebig, such as are also used in determining the gases of the stomach contents. Should we have to deal with ulcerative processes of the rectum, we can obtain the secretion directly upon a glass rod or a platinum loop—best with the use of a speculum and good illumination.

From what has been said, it will be readily seen that the examination of the fæces is divisible into the macroscopical, the microscopical, and the chemical.

1. The Macroscopical Examination

In the last chapter a few striking changes were briefly mentioned and their diagnostic importance dwelt upon. This was necessary because, notwithstanding the best intentions, we can not at the time of the first consultation always obtain test material, and for the time being are compelled to rely upon the statements of the patient. In the following discussion it is intended to give as complete a review as possible of the macroscopical changes of the stools. Consistency and form, appearance, quantity, colour, smell, as well as abnormal ingredients, will have to be considered.

Normally, the fæces are cylindrical in form, with great variations in the calibre of the individual cylinders, or else they are homogeneous, and of a thick, pasty consistency. This difference in consistency depends, among other things, upon the mode of life of the individual and upon the nature of the diet. Under certain conditions variations from the types just mentioned may still be normal.

This fact, often overlooked, is of great practical importance. The following will serve as an example: A patient who has just passed through typhoid fever is constipated. The evacuations occur only after enemata, and resemble the normal fæces of the sheep ("schafkothartig"). In view of the careful diet of reconvalescence, and the lack of exercise which is necessarily associated with a cured case of typhoid, such a condition of the stools is entirely physiological, and it would be a mistake to speak here of atony of the intestines following typhoid. Cases like this occur almost daily, and where constipation and changes of the stool are complained of, it behooves the physician to inquire carefully into the manner of living, and above all into the diet of the patient. Conversely, the occurrence of diarrhœa with a diet consisting for the most part of milk or of milk preparations, or after unaccustomed drinking of sour wines or partaking of too much fruit or sweets, is an entirely normal condition.

Since we have frequently observed false therapeutic measures instituted because of a failure to properly recognise the conditions in question, we believe it best to state these facts, which, without doubt, are known to the experienced physician. Variations in consistency admit of two possibilities: the stool may either be *abnormally* hard and passed in small lumps (scybala, "sheep stool," "hazel-nut" stool), or in long, thin cylinders; secondly, the opposite condition may occur—the stools are passed in a thin, pasty, or even fluid state.

Very hard fæces with occasional furrows (evidently impressions from the tæniæ coli) indicate only long retention within the intestine, and consequent desiccation. Such an appearance speaks for intestinal stricture as little as does the so-called lead-pencil stool. In these cases there are, very probably, spastic contractions of the intestines, such as are often observed in chronic constipation. I would lay more stress, however, upon a different form of stool, which I have frequently seen in stenoses of the intestine. This consists of a homogeneous, thick, pasty, or curdlike evacuation, in which several short cylinders, of the thickness of the small finger, float about. The diagnostic importance of this stool formation must not be overestimated, and only repeated observation of the same condition is of significance.

Thin stools may also vary in two ways: they may be very watery, as in cholera nostras, or asiatica, or they may have a certain admixture of mucus, which can be easily recognised, since it clings to the sides of the glass when the contents are poured out. The microscopical and chemical examinations confirm the presence of mucus.

The *quantity* of stool passed is seldom of any practical importance. In stools of firm consistency the quantity voided is important, if it remain considerably below the normal. It should be remarked, however, that depending upon the nature of the diet (vegetable, animal, mixed, milk, soup, starvation, etc.) the greatest variations occur. There may be repeated small watery evacuations, accompanied by marked tenesmus. Such evacuations indicate inflammatory processes in the lowermost segments of the intestine. Among other conditions, stools of this kind occur very frequently with hæmorrhoids, in acute and chronic dysentery, proctitis, inflammations of the prostate, rectal carcinoma, etc.

The colour of the fæces bears a certain relation to the consistency. Even the normal colour may show variations; thus in purely meat diet it is dark brown (from hæmatin and ferrous sulphate); in a mixed and vegetable diet, although much lighter, it is still brown from urobilin (according to Fleischer[1], also *biliprasin*); it is lightest of all where the diet consists mainly of milk. The longer the stool remains in the intestines the firmer will be its consistency and the darker its colour. Under these conditions it may even assume a tarlike appearance, which I know has led inexperienced persons to believe there have been gastric or intestinal hæmorrhages. Where the intestinal contents pass rapidly through the canal, bilirubin may appear unaltered in the stools; in fact, some stools give bilirubin reaction only. As a result of the absence of reduction of the bile pigments, we frequently find unaltered bile pigments in children's stools. In itself, a green colouration of stools is by no means indicative of bilirubin, for substances containing chlorophyl, when partaken of in large amounts, colour the fæces green. Besides these, there are other alimentary colour changes in the stool that are not without practical significance. Thus the stools are coloured more or less of a brownish red by cocoa, a dark brown with a shade of green by huckleberries and preparations containing them, and also, though not so intensely, by red wine. Iron and manganese salts give to the stools a darker shade than they ordinarily have. According to Quincke[2], in this case the iron is not converted into a sulphate, but, owing no doubt to the action of intestinal bacteria, the iron salts are reduced to iron oxydyl. Similarly, bismuth colours the stools very darkly, though by no means a jet black. As Quincke has recently shown, and as I have been able to confirm through numerous control observations, the cause of this is not the formation of bismuth sulphate, but, like the iron, the bismuth is reduced to bismuth oxydyl. Calomel frequently, though by no means always, imparts a greenish tinge to the stool. According to examinations of Hoppe-Seyler and Wassiljeff, this results from a reduction of a portion of the bilirubin to urobilin. The anti-fermentative action of the calomel depends apparently upon this change. Besides the drugs mentioned there are many others which also cause colour changes in the stools; thus senna, santonin, gamboge, rhubarb and its preparations, colour the stools yellowish.

Acholic stools are very characteristic, and, in view of the phenomena attending their appearance, can not fail to be recognised. Owing to their importance they will be considered in a separate section, to which the reader is referred (see page 103).

The normal *odour* of the stool results from the presence of skatol, and to a lesser degree also of indol. Under pathological conditions the odour depends largely upon the nature of the sickness, and, furthermore, upon the length of time that the fæces have remained within the intestinal canal. As a rule, the longer they stagnate in the large intestine the stronger will be the fæcal odour; conversely, after a rapid passage the odour may be very slight or entirely absent. The rice-water stools of *cholera asiatica and nostras* are the best examples of the last-mentioned condition. In very acute intestinal catarrhs and dysentery the odour may be very slight or entirely absent. In chronic catarrh of the small intestines, too, I have noticed entire absence of any odour. The stools of nursing babies often have a sour and slightly fæcal smell. The evacuations of adults containing large amounts of fat, particularly milk fat, may lack the fæcal odour, but instead take on an offensive smell like fatty acids or even cheese. In amœbic enteritis the fæces have a peculiar *gelatinlike* odour. This was first observed by Quincke and Roos[3], and later confirmed by myself[4].

Closure of the common bile duct is frequently attended by obstinate constipation, and, as a consequence, the evacuations smell very strongly; this condition, however, changes upon the administration of agents which increase intestinal peristalsis. Fetid-smelling evacuations occur in ulcerating carcinoma of the large intestine or of the rectum, etc.

Abnormal admixtures in the stool occur very frequently, and at times may constitute invaluable diagnostic symptoms of the existing intestinal disease.

Blood.—Fresh unclotted blood, when mixed with the stool, must come from the lower portion of the intestines. It will require local examination of the rectum or lavage of the large intestines to determine its exact source. Blood may also appear altered and decomposed, and impart a colour of tar or wagon grease to the evacuations. We have already stated what is most important in this connection on page 64.

Pus.—Pus may appear with the dejections and be recognised by the naked eye. It almost invariably comes from the lower portion of the intestines, for pus from the higher parts, unless voided in very large quantities, is mixed with the fæces, and undergoes physical and chemical changes which make it macroscopically and microscopically unrecognisable.

The appearance of *mucus* in the stools is of special importance. Its significance in the diagnosis of intestinal diseases, and especially of intestinal catarrh, has been taught us by Nothnagel's classical studies[5]. Although in what follows I base my remarks mainly on Nothnagel's teachings, I differ with this clinician in a number of important points, and shall discuss the subject in the light of my own quite extensive experience. In the first place, it must be remembered that in itself mucus represents a normal product, inasmuch as it is always possible with chemical reagents to demonstrate its presence in the stools.* The mucus covering, which is found in very minute quantities partly upon the surface of the fæces and partly mingled with them, is also a normal constituent, being no doubt only a cohesive agent. Hence I can not indorse the statement of Nothnagel, that every macro- and microscopically recognisable admixture of mucus with the stool indicates a change from the actual physiological condition.

Even after a single administration of an active cathartic like castor oil we frequently find a large amount of mucus in the evacuations. We can not here speak of a pathological condition. Furthermore, we know that there is a physiological or, more properly speaking, alimentary constipation and diarrhœa (see above, pages 95 and 96). And here also we can not without definite reason regard mucous admixtures in the stools as pathological. Apart from these restrictions Nothnagel's view is fully correct.

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* On the other hand, Hoppe-Seyler's view that the main ingredient of all fæces, normal as well as abnormal, is mucin, is certainly not correct.

Macroscopically recognisable mucus appears under four different forms:

1. As pure, thick or glistening mucus which is voided as such—i. e., unmixed with the fæces. This points with certainty to a diseased condition of the lowermost segments of the intestines—the rectum, or, at most, the sigmoid flexure. It should, however, be remembered that absence of such mucus does not at all speak against catarrh of the lower intestinal segments.

2. As mucous shreds, or membranes with or without amorphous mucous masses, indistinguishable from that just described. It occurs with any of those intestinal affections so frequently referred to, which we designate as membranous enteritis; these will be spoken of more in detail when we come to the description of this affection in the special part of this work.

3. As tenacious, sticky, gummy, brownish-yellow mucus, intimately mixed with thin pasty fæces. If stirred with a glass rod it adheres in dense, sticky masses, and is separated from the basic mass only with great difficulty.

4. As small shreds of mucus scarcely macroscopically visible. These shreds are best seen by shaking up the fæcal mass in a glass vessel.

With the exception of the variety that accompanies membranous enteritis, and which at times develops in nervous individuals (*colica mucosa*, Nothnagel), all the other forms indicate catarrh of the large intestines.

Although for years I have carefully looked for those frog-spawn or sagolike bodies in the stool whose vegetable origin was first recognised by Virchow, and which are mentioned by Nothnagel in the above investigations, I have thus far never seen them in typical form. Kitagawa, on the contrary, thinks that sagolike bodies of mucous character are met with in enteritis and in intestinal ulcers. Research into this matter is not yet concluded, though if for no other reason than their rarity these bodies can have but slight diagnostic interest.

Mucus in the form fisrt observed and described by Nothnagel as *yellow mucous granules* is barely visible macroscopically. As described by this author, these bodies consist of yellow or brownish yellow or even of dark green granules varying in size from poppy seeds to peas, and having the consistency of butter. Microscopical and micro-chemical examinations have shown that their colour is due to unaltered bilirubin, and it can be readily demonstrated that the ground substance of these bodies consists of mucin or of a body similar to it. According to Nothnagel, *these bodies point to catarrh of the small intestines.* They may occur in the stool singly or in large numbers. Although I make it a rule to examine the stools in every case of intestinal catarrh, I have never seen these yellow mucous granules. One of our best authorities upon mucoid bodies (Ad. Schmidt) also questions their existence and their diagnostic significance. According to Nothnagel, mucus can be recognised by the microscope exclusively, only under the following condition: Where the fæces are formed, are of a firm pasty consistency, and have intimately mixed with them numerous small particles of mucus, which appear under the microscope as small homogeneous, hyaline, grayish-white refractive islets. Nothnagel regards their presence as indicative of a catarrh of the uppermost portion of the large or of the lowermost portion of the small intestine. I have never seen these hyaline mucous "islets" of Nothnagel, and Ad. Schmidt[6], who regards them as dead amœbæ, doubts their mucous structure. At all events, no positive diagnostic importance can be attributed to their presence in the fæces.

Important as is the appearance of mucus in the stools for the diagnosis and location of a catarrh, its absence does not, as I have already remarked, exclude catarrh. For example, as Nothnagel mentions, and I, too, have been able to show in a very typical case (see Part II, Intestinal Catarrh), mucus may be entirely absent in jejunitis. Furthermore, mucus may be entirely absent in intestinal atrophy—so-called lienteritis of old persons. In both these instances the clinical picture is so typical that with careful observation the diagnosis should present no difficulties. Undigested food remnants are visible in the fæces. The opinions even of scientifically educated and experienced physicians differ so widely regarding the significance of this, that I desire to state my own views on the subject.

Per se isolated or occasionally macroscopically recognisable food remnants in the fæces do not indicate disease, for they may result from poor preparation of the food or from insufficient chewing; or else, owing to utter insolubility, these remnants may have entirely escaped the action of the digestive juices. To judge from my own experience, this would apply almost entirely to vegetable substances (fruits, potatoes, legumes, etc.). Macroscopically visible remnants of meat, on the contrary, point to a serious disturbance in the function of the digestive tract, the exact location being only possible through the entire clinical observation, especially in combination with the examination of the stomach contents.*

The constant appearance of large amounts of the above-named vegetables in the fæces points with great probability to anomalies of secretion in the gastro-intestinal canal. In particularly severe cases of chronic intestinal catarrh in which the stomach, as a rule, is also involved, we may regularly find large amounts of undigested meat, as well as of vegetables, fruits, etc., in the fæces. These substances may take on an extraordinary appearance, such as shown in Fig. 12, drawn from a fresh specimen. This important form of enteritis will be again referred to in the special part of this work.

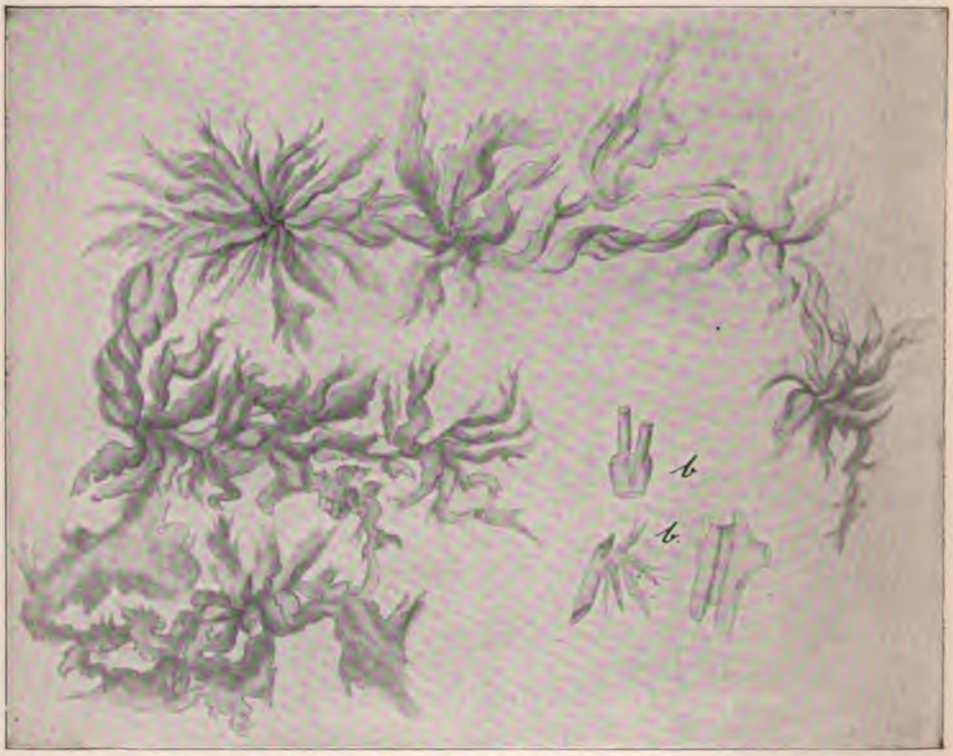

FIG. 12.—SPIRALS OF UNDIGESTED MEAT FRAGMENTS IN FÆCES. (Natural size.) *b*, pieces of bronchi. (Original observation.)

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* [Recently, in a very interesting and instructive paper, A. Schmidt (Deutsche med. Wochenschr., 1899, No. 49) has sought to formulate the principles which govern the appearance of meat remnants in the fæces. By experiments he shows

Occasionally, fragments of new growths (polyps, ulcerated carcinoma, etc.) are seen, although their appearance in the fæces is usually accidental. As is well known, entozoa (ascarides, segments of tapeworm, oxyuris, anchylostomum, trichocephalus, anguillula, etc.) as well as echinococcus are sometimes also found in the stools. The diagnostic importance of these bodies requires no special mention.

2. The Chemical Examination

The practical object of this book prohibits us from describing all chemico-physiological tests. In the following, therefore, we treat only of the more important methods, and at the same time shall discuss the diagnostic importance of each. We shall consider

1. REACTION OF THE FÆCES

The normal reaction of the fæces is neutral or slightly alkaline, changing to feebly acid only when the diet is largely a vegetable one. The reaction becomes very strongly acid on occlusion of the flow of bile into the intestines, being principally due to the presence of fatty acids which have been incompletely or not at all saponified. The qualitative test of the reaction of the stool is made in the usual manner with litmus paper. It should be remarked, however, that the surface of the fæcal masses may give a different reaction than the inner portions. To make a quantitative determination, 20 to 50 centimetres are mixed in a mortar with about ten times their amount of distilled water, and a few drops of $\frac{1}{10}$ phenolphthalein solution or of a good litmus solution are added as an indicator; or litmus paper itself may be used instead. Decinormal NaOH or decinormal $Ba(OH)_2$ (Rubner) are added drop by drop until a neutral reaction

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that the connective tissue is digested by the gastric juice and the muscle fibres and nuclein by the pancreatic juice. The ingestion *pro die* of about 100 grams of finely chopped and lightly fried beef should result in no meat remnants in the stools. From his researches Schmidt concludes:

1. That the appearance under such circumstances of macroscopically visible connective tissue (or of a large amount under a free diet) indicates some disturbance of gastric function (either secretory, i. e., hyper-, sub-, or anacidity, or else motory, i. e., hyper- or submotility).

2. If, in addition to the connective tissue, there are also macroscopically visible muscle fibres present, there must also be a disturbance of intestinal digestion.

3. If visible muscle fibres appear without any connective tissue, there must be a serious disturbance in intestinal digestion, but whether dependent upon anomalies of the pancreatic secretion or upon interference with the secretory or absorptive functions of the intestine itself, it is impossible to state.—TR.]

is obtained. The amount of alkaline solution added to obtain neutralization can be expressed in percentage, as is done with the stomach contents. For example, if it requires 3 centimetres to neutralize 50 grams of fresh fæces, the percentage acidity of the latter will be equal to 6 decinormal NaOH. Conversely, the alkalinity may be determined in suitable cases with decinormal HCl.

As Nothnagel has pointed out, no diagnostic significance attaches itself to the reaction of the intestinal evacuations.

2. DETERMINATION OF ALBUMINOID BODIES IN THE FÆCES

The albuminoid bodies which may occur in the fæces are mucin, albumin, and peptones (albumoses).

(*a*) *Determination of Mucin*

Either the fæces themselves or some mucoid masses mixed with them may have to be analyzed. In the first instance the fæces should be rubbed up with water, and an equal volume of limewater added; after the mixture has stood for several hours the filtrate is to be tested for mucin. Where mucoid bodies themselves are to be analyzed, they should first be dissolved in weak potassium or sodium-hydrate solution and then tested with acetic acid. Insolubility in an excess of the acetic acid would speak for mucin. To avoid confusion with mucinlike nucleo-albumin, the precipitate must be further tested by boiling with dilute mineral acids. Should a substance which reduces oxid of copper be readily thrown down, the precipitate may be regarded as being mucin. It must, however, be shown that, after repeated reprecipitation, the substance in question is still free from phosphorus.

A much simpler and an equally effective test, in my opinion, is the macroscopical staining of the mucus by means of Ehrlich's triacid solution. A small piece of mucus is broken up in sublimate alcohol and allowed to stand a short time; the sublimate solution is then replaced by distilled water, and a few drops of triacid solution added. If the fragments are coloured green, they are composed mostly of mucus, and if red, there is an excess of albumin (Ad. Schmidt[6], Pariser[7], J. Kaufmann[8]). The diagnostic importance of mucus has already been dwelt upon.

(*b*) *Determination of Albumin*

In testing for albumin in the fæces, the latter are to be extracted with a large amount of water to which a trace of acetic acid has been added, and the watery extract filtered a number of times. The filtrate should then be tested with the same reagents as used in testing the urine for albumin; of these, acetic acid and ferrocyanid of potash are particularly to be recommended. For the quantitative determination of the albumin, Magnus Blauberg[9] recommends the following procedure: 3 to 5 grams of dried fæces are repeatedly (three or four times) digested in ten times their volume of thymol water * (each time for three or four hours), and the clear supernatant fluid filtered each time through the same filter. To these combined filtrates are added one half their volume of a saturated solution of sodium chlorid and an excess of Dragendorf's tannin mixture (tannin, 20 grams; glacial acetic acid, 37.5 grams). After a while the filtrate that forms is brought upon a previously washed filter, then dried, and the amount of nitrogen determined by the well-known Kjeldahl method. In typhoid diarrhœic evacuations and in two cases of acholic stools von Jaksch[10] found demonstrable quantities of albumin.

(*c*) *Determination of Albumoses and Peptones*

Von Jaksch[11] employs the following method: The fæces are mixed with water until they have acquired a thin, pasty consistency; the mixture is then boiled, filtered while still hot, and the clear filtrate tested for albumin with acetic acid and ferrocyanid of potash. Usually a slight cloudiness follows the addition of the acetic acid (mucin), and does not increase upon addition of the potassium ferrocyanid. If such be the case, the mucin must be precipitated by a solution of acetate of lead and the filtrate further treated with phospho-tungstic acid. The fluid which finally remains should be tested for the biuret reaction (caustic soda and copper sulphate). If, after the boiling, albumin is still present, it must be removed by combining it with acetic oxid of iron, and we then proceed in the above-described manner. Magnus Blauberg[9] simply precipitates the combined watery extracts of the fæces with phospho-tungstic acid, collects the precipitates which he dilutes with water, and then carefully floats caustic soda and very weak solution of copper sulphate upon it (biuret test). To separate albumoses from peptones, the former should be removed by "salting" them out with ammonium sulphate.

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* Made by shaking distilled water with a saturated alcoholic solution of thymol for a long time, and then filtering.

In normal stools von Jaksch never found peptones. In pathological stools he found them under most varying conditions—frequently in typhoid fever, and wherever much pus was produced (dysentery, tubercular ulcers, and peritonitis, with rupture into the intestines). In affections of the liver the relations were extremely varied. In the stools of nurselings Blauberg almost constantly found protein substances.

3. DETERMINATION OF CARBOHYDRATES IN THE FÆCES

Hoppe-Seyler[12] recommends the following procedure for the determination of carbohydrates: The fæces are distilled, the residuum extracted with alcohol and ether, the extract boiled with water, then filtered, the filtrate partially evaporated, and then tested for reducing substances by boiling with dilute sulphuric acid, supersaturating with caustic soda, and then adding sulphate of copper solution. If we wish to test for sugar, the fæces should be extracted with water, the albuminoid bodies in the extract precipitated with acetate of lead, the solution freed from lead by means of a current of CO_2, then filtered, and the filtrate tested for sugar by Trommer's or Nylander's test, etc. The quantitative determination of sugar is made with dried fæces, after the manner of Allihn and Liebmann.

It has been already stated (page 38) that the carbohydrates are more completely digested and utilized than any other variety of nutritive substances. This accounts for their being so constantly absent (at least in large quantities) from the fæces. In numerous examinations of watery extracts of the fæces, I have only twice obtained a distinct Trommer reaction; with Lugol's solution I have never obtained a reaction.

4. DETERMINATION OF FATS IN THE FÆCES

For the determination of fat it will, as a rule, be sufficient to shake the fæces with large amounts of ether until nothing more can be extracted, and then to evaporate the ether over a water bath. The ether takes up only the neutral fats and the fatty acids, but not the soaps. The latter are demonstrated by splitting up with acids and then extracting with ether. For the quantitative determination of the total amount of fat in the stools as should be made, and as are indispensable in careful examinations of the so-called "fatty stools," we must first estimate the amount of the neutral fats and the fatty acids, then the fatty acids alone, and from the difference we learn the amount of neutral fats. Finally, through the agency of acids, the fatty soaps in the remainder of the first portion are split up into fatty acids, and as such are directly estimated. It would lead too far to enter into a detailed description of the methods involved in these examinations. To those who wish to better acquaint themselves with them we recommend Fr. Müller's Studien über Icterus, Zeitschrift für klinische Medicin, Bd. xi, S. 45–113, and von Noorden's Beiträge zur Lehre vom Stoffwechsel, Heft 1, S. 109 u. f., Berlin, 1892.

In addition to the fixed fatty acids, volatile ones—succinic, butyric, propionic, acetic—also occur. They are isolated, and determined by fractional distillation. For details the reader is referred to the works of Hoppe-Seyler—Thierfelder, Hammarsten (Handbuch der Physiologischen Chemie), and von Jaksch (Klinische Diagnostik, p. 280). No diagnostic significance attaches itself to the determination of the volatile fatty acids.

Normally, fat, especially when taken in large quantities, may appear with the fæces. It is met with partly as free fat, in the form of fatty acids, and partly as soaps of calcium and magnesium. A good example of the limitation of fat assimilation is found in the taking of large quantities of olive oil, such as are at present employed in cholelithiasis. In these cases we may frequently find in the fæces fat in lumps varying from the size of peas to hazel nuts; but when taken in a form difficult of solution (pork, mutton, tallow, etc.), it will appear in the dejections even though the amount partaken of be small. We speak of *fatty stools* only when the fæces are so loaded with fat that this is even macroscopically visible. This variety of stools has a light silver-grayish appearance, and if allowed to stand in the cold, especially after being previously stirred, two layers—a lower waxy yellow and a fæcal one—will form. In other and less marked cases we will still be able to recognise fatty particles or yellowish translucent fatty plaques, which may be isolated for further examination.

A priori, it is evident that fatty stools may result from two causes: either an incomplete absorption of fat well split up and saponified, or an insufficient splitting up, whereby its absorption into the lymph and chyle channels is rendered difficult. Finally, both causes may act simultaneously. There can be no doubt whatever as to the occurrence of the first possibility. Biedert[13] and Demme[14] have shown this in catarrh of the small intestines in children, and Fr. Müller[15] in adults with caseation of the mesenterial glands. According to Nothnagel,* fatty stools occur also in atrophy, amyloid disease, and tuberculosis of the intestines.

It is more difficult to explain the second cause of fatty stools—i. e., anomalies in the splitting up of the fat which result from pancreatic disease or from insufficient excretion of bile. Although the investigations of Abelmann[16] and Minkowski have shown that in animals after extirpation of the pancreas absorption of fat no longer occurs, control investigations of Sandmeyer[17] and Teichmann[18] have also shown that, in spite of exclusion of the pancreatic secretion, absorption of fat will still take place. In two cases of complete intestinal obstruction, and in one case of cystic degeneration of the pancreas where large amounts of fat had been given, Fr. Müller[15] could find no increase of fat in the stools.

Absence of pancreatic juice, therefore, does not prevent the digestion of fat. On the other hand, the careful and thorough examinations of Fr. Müller have shown that in these cases an insufficient splitting up of fat occurs. Whereas with a normal secretion of pancreatic juice or in icterus the greater portion (84 per cent) of fat in the fæces was split up, only 40 per cent (in round numbers) was split up in the absence of the pancreatic juice. Should this fact be confirmed, it would constitute an important sign for diagnosing disturbances of the pancreatic secretion or occlusions of the pancreatic duct. This would be all the more important, since the other symptoms of these conditions—mellituria or maltosuria, multiple muscle fibres in the stool, and ptyalism—occur very rarely, and are only conclusive when all three are present. Conversely, since thorough splitting up of fat may result from bacterial action, it does not at all speak against occlusion of the pancreatic duct. Very much interest also attaches itself to those acholic stools without icterus, which Bamberger[19] and Gerhardt[20] described, and which have been particularly studied by Nothnagel[21], von Jaksch,† Berggrün, and Katz[22] and Pel.[23] I have observed five cases of this kind. These acholic stools without icterus are characterized by containing a large percentage of fat or of fatty acids. Their reaction, therefore, is strongly acid. Urobilin has been repeatedly found in these colourless stools (in one of my own cases also). In the majority of cases the acholia was only temporary. In one case under my observation the acholia continued for fourteen days; unfortunately, further observation of the case was impossible. The large amount of fat present is very probably due to insufficient absorption. It is unnecessary to repeat that the rice-water stools of cholera nostras and asiatica are also acholic; the cause is very probably an entire absence of intestinal secretion.

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* Nothnagel, loc. cit., p. 20.

† Von Jaksch, loc. cit., p. 292.

The class of acholic stools under discussion has been observed under the most varied conditions in severe as well as in light cases. Thus Nothnagel observed acholia in leukæmia, in cancer of the stomach and intestines, in simple intestinal catarrh of children and adults, and most frequently of all in advanced cases of pulmonary tuberculosis. Von Jaksch found acholic stools in intestinal tuberculosis, chronic nephritis, anæmia, and scarlet fever. Berggrün and Katz observed and ascribed great importance to them in tubercular peritonitis in children. I myself have found acholic stools in cholelithiasis, where the icterus appeared a few days later. I have also seen it in a very feeble patient who suffered from stricture of the esophagus, and after the use of Carlsbad water and salts in a young lady suffering from chronic catarrh of the large (and small ?) intestines; furthermore, in a patient with cancer of the pancreas, and finally in a severe case of chronic constipation, where, on account of other clinical manifestations, the probable diagnosis of cancer of the large intestines had been made.

At present our ideas as to the causes of this form of acholia have a purely theoretical basis. Nothnagel believes it is occasioned by a temporary cessation of biliary secretion. Von Jaksch thinks the lack of colour is due to the non-formation of urobilin from bile pigments, or else that colourless products of decomposition of bilirubin, "leukourobilin" (von Nencki), are formed from the bile pigments. The absence of colour is very probably due to a large percentage of fat. We can readily imagine that, owing to certain influences (at present unknown), the fat, or rather the fatty soaps, are only partially or not at all absorbed from the small intestines. On the other hand, it can not be denied that the abnormal excretion of fat from the intestines may be caused by temporary or permanent disturbances of pancreatic or biliary secretions. Owing to the numerous possible causes of this form of acholia we can not at present assign any great diagnostic value to it.

5. Determination of Blood and Blood-colouring Matter in the Fæces

With fresh blood, such as comes from the lower portion of the intestines, the macroscopical appearance of the fæces is so characteristic that a microscopical control examination will seldom or never be required. Well preserved red and white blood-corpuscles may be demonstrated. Where the coloring matter of the blood has undergone changes the condition is quite different. The presence of blood-colouring matter may be demonstrated by microscopical, chemical, and spectroscopic analyses, which will now be collectively considered.

(*a*) Blood, when present in the fæces, can seldom be demonstrated by *microscopical examination*. Even where the stools were still intensely red coloured, Nothnagel never could find erythrocytes in the fresh hæmorrhages of typhoid fever. In these cases, however, we can observe clumps of pigment which, according to von Jaksch,* are composed of hæmatoidin.

(*b*) *The Micro-chemical Demonstration* (*Hæmin Test*).—A small particle of fæces that appears to contain blood-colouring matter is dried, then pulverized, and together with a minute particle of common salt and one drop of glacial acetic acid brought upon a glass slide, and the whole mixed carefully for a few moments. A cover glass is then placed over the mixture and glacial acetic acid allowed to flow under the cover glass until the latter uniformly touches the slide. Thereupon the slide is held over a very small flame, care being taken that the glacial acetic acid does not reach the boiling point; the heating may be repeated with the addition of fresh glacial acetic acid. When the specimen has cooled it is examined with the high power for hæmatin crystals.†

(*c*) *Chemical Demonstration* (*Weber's Test*[24]).—The fæces are extracted with glacial acetic acid and ether, and to a few cubic centimetres of this extract 10 drops of the tincture of guaiac and 30 drops of oil of turpentine are added. In the presence of hæmatin we can observe a pretty blue or violet colour, which, after the addition of water, can be extracted with chloroform.

(*d*) *Spectroscopic Demonstration.*—A portion of the most darkly coloured part of the fæces is stirred with a little water, and, after the addition of a few drops of concentrated acetic acid, shaken with one fifth its volume of ether; after a few moments the ether separates, and, if the fæces contain blood, is coloured a brownish red (due to the hæmatin). The ethereal hæmatin solution shows four spectroscopic absorption bands: one in the red, a second in the yellow, a third between the yellow and the green, and a fourth between the green and the blue. That in the red is by far the darkest and most clearly defined.

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* Von Jaksch, loc. cit., p. 232.

† For unknown reasons the hæmin test with fæces does not always succeed.

In most cases the demonstration of blood in the fæces is easy, but the determination of its origin is more difficult. In the first place, we must remember that blood may be physiologically introduced into the fæces through raw meat and some varieties of sausages. If these can be excluded a large amount of blood in the fæces is always a pathological condition. The causes are so numerous and varied that only through general and local examinations can the source be determined. The diseases of the intestines which frequently or occasionally cause hæmorrhages include ulcers, neoplasms, hæmorrhoids, amyloid disease, intussusception, stenosis, ileus, anchylostomum, bothriocephalus, trichocephalus dispar, etc.

6. DEMONSTRATION OF BILIARY MATTER IN THE FÆCES (BILE PIGMENTS, BILIARY ACIDS)

As previously mentioned, the brown colour of normal stools is due to colouring substances formed through reduction of the bilirubin or of the biliverdin of the bile. In adults, unaltered bile pigment (bilirubin, biliverdin) appears under pathological conditions only. Hence the demonstration of normal as well as of abnormal fæcal colouring matter may have some diagnostic value.

(*a*) *Demonstration of Bile Pigments in Toto* (*Huppert's Test*)

Thin fluid fæces (or artificially thinned fæces) are placed in a flask, an equal volume of milk of lime is added, and after repeated shaking the mixture is poured upon a small filter; the precipitate which remains upon the filter consists of hydrobilirubin lime. This should be washed, and, while still moist, placed into a boiling flask with a very long neck, together with 20 cubic centimetres of alcohol to which sulphuric acid has been previously added to distinct acid reaction. The mixture is heated to boiling; in the presence of biliary colouring matter the fluid will acquire an emerald green or a bluish green colour. Positive reaction with Huppert's test simply indicates the presence of bile pigment in the fæces, but tells nothing of its nature.

(*b*) *Test for Urobilin*

1. Mehu's test. The fæces are extracted with water and sulphuric acid in proportion to 2 grams to the litre and ammonium sulphate in substance are added to the watery extract. The resulting precipitate is separated by filtration, washed with a warm saturated solution of ammonium sulphate, dried over a water bath, and then extracted with hot ammoniated alcohol. Spectroscopic examination of this extract shows an absorption band between B

and F. The precipitate may be otherwise treated; it may be dissolved with ammoniated water and a 10-per-cent solution of chlorid of zinc added. In the presence of urobilin the fluid will show a very pretty fluorescence.

2. A. Schmidt's test.[25] With a glass rod a fragment of fæces about the size of a bean is rubbed up in a beaker or porcelain dish with a concentrated watery sublimate solution. If urobilin be present the mixture immediately, or after a short time, acquires a rose colour; or if biliverdin be present, a green colour. According to the experiments of Dr. Hari in my laboratory, the reaction can be more quickly obtained by shaking the fæces with concentrated sublimate solution, filtering, and then adding chloroform to the filtrate. In the presence of urobilin a very pretty rose colour appears at the point of contact of the two fluids.

3. Fleischer's method.[26] If a small quantity of fæces be placed in a small flask or a test tube and alcohol (to which hydrochloric, sulphuric, or acetic acid has previously been added) be poured over it and the whole allowed to stand a short time, it will be coloured yellowish or brownish, according to the proportion of urobilin contained. If the alcohol be then poured off and a few drops of caustic soda or ammonia solution added, and then chlorid of zinc solution, a green fluorescence with direct light and a rose-coloured or yellowish with transmitted light will be obtained. The degree of colour reaction is proportionate to the amount of urobilin present in the stools. If ammonia solution be added to a watery extract of the fæces, and, after filtration, chlorid of zinc solution, we will obtain a beautiful rose red or a dark red precipitate in the presence of urobilin; in the absence of urobilin, however, this precipitate will remain uncoloured. If this precipitate is brought upon a filter and washed with ammoniated alcohol, we will obtain a more or less marked greenish fluorescence.

(c) *Determination of Unreduced Bile Pigments (Bilirubin, Biliverdin), Gmelin's Test*

If nitroso-nitric acid be added to the fæces, the characteristic colours—red, violet, and green—will appear (in most cases only a green colour is seen). In like manner, after filtration of the fæces, we can obtain this test with the dried filter paper. In order to demonstrate bile pigment micro-chemically, a drop of the same acid is added to a small particle of fæces placed upon a glass slide. The reaction can be obtained either with the entire fæces or else with some of the ingredients—viz., mucus, epithelium, muscle fibres, etc.

(d) *Determination of Bile Acids*

A small quantity of the fæces is extracted with alcohol, the latter evaporated, and the residue dissolved in a very weak watery solution of soda. Upon addition of cane sugar and a few drops of sulphuric acid the characteristic red and purple colours of the bile acids appear (Pettenkoffer's test). Since the greater portion of bile acids normally disappear by absorption, their presence in the dejections is always pathological.

The diagnostic significance of the tests for biliary pigment in the fæces lies mainly in the fact that their presence proves a completely unobstructed flow of bile into the intestines, while their absence speaks for an obstruction to the flow of bile. According to Nothnagel,[5] normal biliary pigment can be demonstrated only in thin, pasty, and fluid evacuations by Gmelin's test. If in such evacuations distinct reaction for biliary pigments is obtained, and the biliary pigment clings to the mucus or to the cylindrical epithelium, it points to catarrh of the small and of the large intestines. With a well-marked reaction we are justified in assuming that catarrh of the upper part of the small intestine is also present.

7. Absence of Bile Pigment (Acholia) and Colourless Stools

Whenever there is an obstruction to the flow of bile, colourless, clay-coloured stools result; also absolutely colourless stools may occur when there is no obstruction in the biliary passages. In obstruction to the flow of bile lack of colour is, on the one hand, due to an absence of bile pigments, and on the other to the presence of abnormal amounts of fat and fatty acid in the stools.

Bunge[27] has pointed out that the clay colour is due not to the absence of bile pigment but to the presence of large amounts of fat. If we extracted such acholic stools repeatedly with ether, they would assume a brownish colour, which, according to Bunge, results from the presence of hæmatin and sulphate of iron. Guided by this, Fleischer[26] examined for and actually found hæmatin in the three acholic stools; in a fourth he did not succeed. In all four stools urobilin was entirely absent. From these investigations Fleischer hopes for a better understanding of the causes and the nature of acholia in the fæces.

Acholic stools occurring simultaneously with icterus point to an affection of the liver, the nature of which can only be determined by a closer examination.

8. Determination of Aromatic Substances in the Fæces (Phenol, Indol, and Skatol)

These aromatic bodies occur normally as well as pathologically.

(*a*) *Phenol.*—To demonstrate the presence of phenol a portion of fæces must be distilled, and the distillate made alkaline with caustic potash and again distilled. The phenol then remains, and is purified by distillation with sulphuric acid. By the addition of bromin water it can be demonstrated in the distillate as tribromophenol. Upon heating the distillate with Millon's reagent a red colour is obtained. A violet colour is obtained by the addition of chlorid of iron.

(*b*) *Indol and Skatol* (Brieger).—To separate these bodies from phenol, the distillate of the fæces, after being made alkaline, is again distilled. Indol and skatol then pass over with the vapour of water. Indol forms colourless scales, and is soluble in hot water and in alcohol. Skatol also forms colourless scales, but is soluble with much more difficulty in water, and has a disagreeable penetrating odour. Unlike indol, it is not decomposed by caustics. With nitroso-nitric acid indol gives a distinct red colour; when more concentrated a red precipitate may result. Pine shavings moistened with muriatic acid are coloured red in a short time by an alcoholic solution of indol. Skatol gives neither the first nor the second test. At present these substances have no diagnostic significance, but a suitable method for their quantitative determination would very probably enable us to recognise different degrees of putrefaction within the intestines.

9. Determination of Ferments in the Fæces

For the purpose of demonstration, the ferments may be extracted with glycerin, and the glycerin extract used; or, what is simpler, the fæces may be stirred with thymol or chloroform water, then filtered, and the tests made with the filtrate. For fæces, Leo[28] also recommends the use of blood fibrin, which has the property of absorbing ferments (discovered by Wittich). For this purpose fæces are mixed with chloroform water until they form a thin, pasty mass. In this mixture we suspend 2 to 5 grams of finely divided, previously boiled blood fibrin, which is inclosed in a gauze bag tied with a thread. After twenty-four hours the gauze bag is taken out of the fæces, the fibrin washed a number of times with water, and then tested for the various ferments. To test for diastase, a small piece of fibrin is placed with some thin starch paste in an incubator, and after a while is tested with diluted Lugol's solution for the presence of sugar or the disappearance of starch.

To test for trypsin, a few pieces of fibrin in a 1-per-cent solution of soda are placed for a while in the incubator, and then tested for albumoses with caustic potash and very dilute sulphate of copper solution. If albumoses are present a pretty rose-red [or onion-red] colour is produced (biuret reaction). Von Jaksch[29] and Leo[28] have demonstrated the presence of ferments in the dejections; diastase and invertin were found in children by von Jaksch, in adults by Leo. In normal and in many pathological conditions Leo was unable to find tryptic ferments, but he did, however, find them in many cases of diarrhœa. In a case of jejunal diarrhœa I found both amylolytic and tryptic ferments.

10. DETERMINATION OF BILIARY GRAVEL AND GALL STONES IN THE FÆCES

As is well known, biliary gravel and gall stones very often appear in the stools after attacks of cholelithiasis. They are very readily recognised in some cases, particularly where the stones are of large size, or are passed in large numbers. On the other hand, the macroscopical recognition of biliary gravel is more difficult. Cholesterin is the principal ingredient of these concrements. In addition, they contain bile pigment combined with calcium (bilirubin-calcium), and bile acids combined with calcium, calcium soaps, and the carbonate of calcium. Appearance, colour, consistency, and size of the stones vary according to the proportion of the different ingredients. We differentiate especially between cholesterin stones which are hard and concentrically formed, and dark pigment stones which are not concentric, and are soft, small, and composed principally of bilirubin calcium.

Chemical Examination.—This includes principally the demonstration of cholesterin and biliary pigments. The concrements are pulverized in a mortar, and in order to remove all biliary components are boiled in water. After drying, the residue is extracted with a mixture of equal parts of alcohol and ether. This extract is then poured off, evaporated over a water bath, and under the microscope shows cholesterin in large rhombic plates with step-like notches. Chemically, cholesterin can be demonstrated by dissolving a portion of the residue in chloroform and adding an equal amount of concentrated sulphuric acid. The chloroform solution then shows the following play of colours: blood-red, purple-red, and after longer exposure to the air, blue, green, and finally yellow. The micro-chemical demonstration of cholesterin is still easier. If to the crystalline mass on a glass slide concentrated sulphuric acid be added, the edges of the crystals will assume a carmin colour, which upon the addition of Lugol's solution changes to violet.

To demonstrate gall-stone fragments we add dilute sulphuric acid to the residue left after boiling with water (*vide* above). The formation of gas shows the presence of a carbonated salt. The mixture is heated, and after cooling is extracted with chloroform. With this chloroform extract Gmelin's test (see page 108) is to be made.

For further tests for the various ingredients of bile concrements, the reader is referred to text-books on physiological chemistry.

11. Pancreatic Stones

Pancreatic calculi are very rarely passed in the stools. These stones have a rough surface, are friable, and can be broken off in facets. They are readily soluble in chloroform, and, according to Minich,[30] when heated evolve an aromatic odour. In one of Leichtenstern's[31] cases chemical examination showed only carbonate and phosphate of calcium, but no bile pigment or cholesterin. The finding of pancreatic stones is of considerable diagnostic interest, for it proves conclusively the existence of a calculus. Unfortunately, owing to their great friability we rarely find these stones. We would, however, recommend that the dejections be examined in cases of cholelithiasis without icterus, and particularly in cases of the so-called "neuralgia of the liver."

12. Fæcal Stones (Coproliths) and Intestinal Stones (Enteroliths)

Fæcal stones or coproliths are formed from inspissated masses of fæces and may acquire a size and firmness sufficient to cause obstruction of the intestine. They develop in portions of the large intestine in which the outward propulsion of the contents is attended with difficulty—i. e., at the flexures and in the ampulla of the rectum. As is well known, the vermiform appendix is a special point of predilection for fæcal stones (see chapter on Appendicitis, in Part II). Real enteroliths, on the other hand, are much smaller bodies, and only very seldom give rise to intestinal obstruction. According to Leichtenstern's investigations, we may distinguish three forms of enteroliths, viz.: 1. Heavy, brown, concentric concrements of stony consistency, composed mainly of phosphates of calcium and magnesium, and containing some foreign body in their interior (e. g., pieces of bone, hairs, fruit seeds, ascarides, ova, etc.). 2. Stones light in weight, composed of a fungoid mass of undigested vegetable matter. They occur in poor people who eat much oatmeal (oat stones). 3. Stones resulting from long-continued use of certain drugs (calcium carbonate, magnesia, sodium bicarbonate, salol, alcoholic solutions of shellac, etc.). This variety may acquire a considerable size. For the chemical character of these stones the reader is referred to text-books on physiological and pathological chemistry.

13. Determination of Inorganic Substances in the Fæces

The fæces contain inorganic salts in amounts varying from 1 to 8 per cent. These consist principally of earthy phosphates; in addition, there are also small amounts of iron, silicic acid, sodium chlorid, sodium sulphate, etc. Of the soluble inorganic substances, sodium chlorid, at all events, possesses practical interest. To demonstrate its presence, the fæces should be mixed with water and allowed to stand a few hours and then filtered. The filtrate is acidulated with nitric acid, and if a cloudiness results should be again filtered. Thereupon a few drops of silver nitrate are added and a white precipitate forms (silver chlorid). For the quantitative determination of sodium chlorid, see the text-book of Hoppe-Seyler and Thierfelder.[12]

3. The Microscopical Examination

Technic.—Thin, pasty, or fluid fæces may be examined without special preparation.* For the better examination of the insoluble portions, fluid fæces may be poured into a conically shaped glass, or, still better, centrifuged (Herz[32]). In the case of firm stools, a small particle is taken and mixed with physiological salt solution. For very exact examinations it is best to take only particles the size of a pin's head. The ingredients to be microscopically examined for are:

1. Food remnants.
2. Elements derived from the intestines.
3. Micro-organisms.
4. Crystalline bodies.

1. The following food remnants are met with normally or pathologically:

(*a*) *Muscle Fibres.*—These occur not only in excessive meat diet, but also in mixed diet. They may have well-preserved striæ, or more frequently they are seen as prismatic bodies with indistinct or obliterated striæ. Nothnagel,[21] however, states that with the very high power even in these cases he could observe transverse striations. Occasionally the muscle fragments can be readily recognised macroscopically, or even singled out as brown spots. Under most diverse conditions muscle fibres are found in increased numbers; in diabetics, who, as is well known, eat very much meat, in persons suffering from fever, particularly when there is increased peristalsis (e. g., typhoid)—in fact, in any condition in which there is a marked increase in the peristalsis of the small and large intestines. In general the appearance even of large numbers of muscle fibres in the fæces has no diagnostic significance. Only their constant increase has any definite value. In the following sentence Nothnagel very carefully formulates this law: "Only where, in the absence of fever and in the presence of definite symptoms of catarrh (mucus, etc.), we find an abnormally large amount of muscle fibre in the fæces, is it permissible to conclude that catarrh of the small intestines is very probably also present."

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* The addition of 1- or 2-per-cent formalin solution effectively deodorizes fæces.

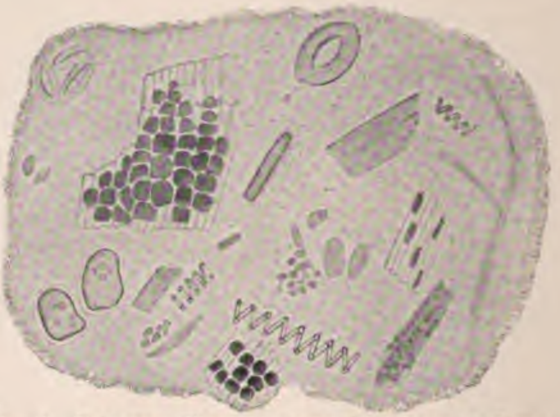

FIG. 13.—DIFFERENT VEGETABLE SUBSTANCES FOUND IN FÆCES.
(Original observation.)

I fully agree with this statement, and would complete it by remarking that the macroscopical rather than the microscopical examination is decisive. Should we constantly observe undigested masses of meat, together with large quantities of mucus in the fæces of patients under moderate meat diet and with well-preserved teeth, the diagnosis, catarrh of the small intestines, is almost certain. The reverse of this statement is not true. Although it is true that in pancreatic disease large quantities of muscle fibres frequently appear in the stools, we may to-day regard as entirely disproved the opinion defended by Friedreich[33] a number of years ago, viz., that abundance of muscle fibres in the fæces indicates pancreatic disease.

(*b*) *Starch.*—Normally, and also in disease, starch is so completely digested that the appearance in the fæces of numerous vegetable remnants, and especially of well-preserved, concentrically arranged starch granules, is of pathological significance. According to my investigations, the following differences have been noted: Upon the addition of iodin to the fæces, in many cases a blue colour is obtained, while in others again a more violet or even a rose colour appears. Without doubt the first colour (blue) is indicative of a more serious interference with starch digestion. Besides isolated starch granules we may also find vegetable remnants that contain starch, and which appear either as threads, particularly spirals, or as large, square, meshlike bodies (Fig. 13) frequently containing chlorophyl, or, finally, as large striated globules similar to those that I have described and pictured in the stomach contents.[34] It is very remarkable that starches, unlike muscle fibres and fats, are never stained by bile pigment.

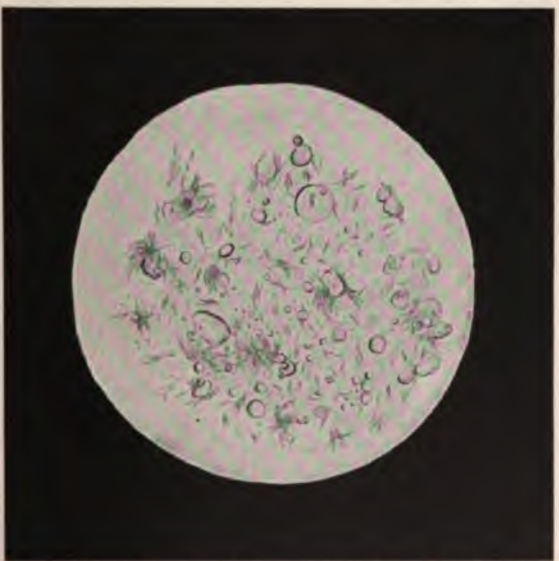

FIG. 14.—FATTY STOOLS, SHOWING A LARGE AMOUNT OF FATTY ACID CRYSTALS. (Original observation.)

(*c*) *Fats.*—Fat appears either in the form of drops or of colourless, occasionally yellowish, clumps. Fat crystals consist either of fatty acids or of fatty soaps. Fatty acids (Fig. 14) are short, delicate, partly curved needles, whereas the soaps (Fig. 15) are formed as long needles arranged in clusters or fan-shaped. When heated over a flame the fatty acids dissolve, but the soaps remain unchanged. Fatty acids are soluble in ether; soaps are only soluble in ether after previous splitting up by acids. These characteristics enable us in every case to determine the nature of fatty crystals.

Rieder[35] has recently recommended a new colour reagent for fats, suitable for clinical purposes. It is known as Sudan III, and is a diazo-colouring substance with the formula $C_{22}H_{10}N_4O$. It is employed in concentrated alcoholic solution, filtered just before using. In fatty stools the fat drops are stained from an orange to a blood-red colour, while fatty acid needles and calcium and magnesium soaps remain uncoloured.

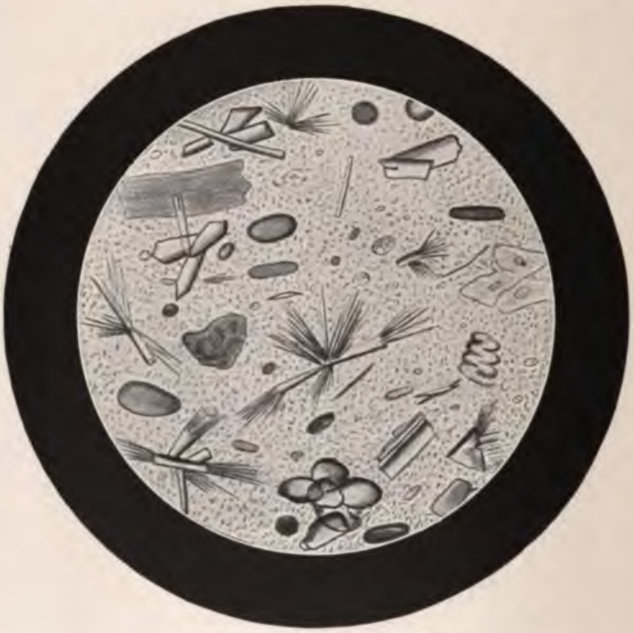

FIG. 15.—FATTY SOAPS IN FÆCES.
Taken from a case of chronic catarrh of the small and large intestines.
(Original observation.)

(*d*) *Coagulated Albumen.*—In diarrhœal stools, especially after excessive dieting with milk, we may, according to Nothnagel, see lumps of casein varying in size from half a lentil to a pea, and frequently stained externally with bile, while internally they are as white as milk. In like manner, after eggs have been partaken of in large quantities, small fragments of them may be observed in the dejections.

2. The substances derived from the intestines themselves include red and white blood cells (pus cells), epithelium, mucus, and tumour fragments.

(*a*) *Blood and Pus Cells.*—Regarding the red blood cells I would refer the reader to what has been already said on page 106. Although exceedingly rare, white blood cells do appear normally in the stools; they are more frequently met with in pathological stools, and are most frequent in catarrhs. Their appearance in great numbers indicates ulcerative processes within the intestinal canal. The most important facts concerning this subject have already been considered on pages 64 and 95.

(*b*) *Epithelium.*—This is usually of the cylindrical and but seldom of the pavement variety. Very rarely in diseases of the rectum (rectal cancer, proctitis) we find pavement epithelium. It is, however, of no symptomatic importance. Cylindrical epithelium is more important. It may be unaltered, or more frequently can assume the greatest alterations in form. The cells may or may not retain their nucleus (degenerated epithelium). Microscopically we may observe all possible changes from the normal appearance of epithelium to that of degeneration. Minute fat globules are also frequently seen in the epithelial cells. A. Schmidt[25] has indisputably proved that mucous shreds, too, may contain a considerable amount of fat. Fig. 16 explains all this much better than any description could. Epithelial cells are best studied in fresh mucus, such as is obtained in test lavage. In a few instances I have also noticed goblet cells, occasionally with intact basement membrane. Large quantities of cylindrical epithelium with mucus usually indicates a desquamative catarrh, whose location must be determined by other symptoms and signs, some of which have already been mentioned. In addition to these formed structures in normal and pathological fæces, we may also find large and small bodies that can be differentiated with difficulty and that are extremely resistant to reagents. This detritus is derived in part from the food as well as from the secretions and excretions of the intestines.

(*c*) *Mucin.*—Mucin is mainly characterized by its striated basement substance, in which epithelial cells are embedded in varying numbers. In some cases the cells predominate to such an extent that the striated structure of the mucin becomes indistinct or is apparently absent. Still, in some portions of the specimen it will always be recognisable by its layerlike appearance. In doubtful cases its presence can be determined by the Hoyer-Ehrlich thionin colour test. Mucus is here coloured a reddish violet, and all other proteid bodies blue. According to Sven Akerlund,[36] eosin, safranin, methylene blue, and hæmatoxylin also colour mucus very satisfactorily, but not so as to admit of any differentiation from other proteid bodies. We have already discussed the significance of mucus in the fæces (page 95). We would, however, repeat that pure, macroscopically visible mucus, such as is obtained in intestinal lavage, can only come from the large intestines.

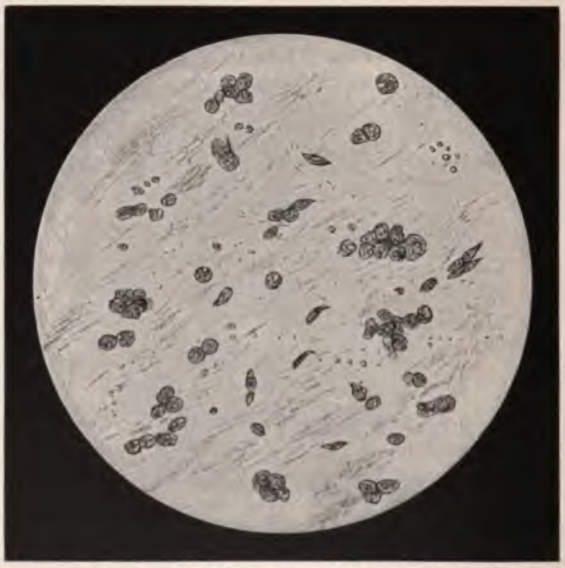

FIG. 16.—NORMAL AND DEGENERATED EPITHELIAL CELLS FROM THE MUCOUS SHREDS OF A CASE OF MEMBRANOUS ENTERITIS. (Original observation.)

3. *Micro-organisms.*—The fæces contain a greater number of micro-organisms than any other excretion of the body. They certainly play a great physiological and pathological rôle. Notwithstanding that the important investigations of Nencki, Macfadyen, and Sieber,[37] as well as of Thierfelder and Nuttal,[38] have shown that normal digestion is possible without the aid of bacteria, we can not deny them some significance. Their activity need not be limited to action upon digestive matter, but may also include reciprocal action on other pathogenic organisms. As usual in bacteriology, we here distinguish moulds, yeasts, and schizomycetes (bacteria).

(*a*) I have but once observed mould fungi in fæces (aspergillus filaments). The oidium albicans has occasionally been observed in the stools of children.

(*b*) *Saccharomyces.*—Of these the yeast is the most important. It is a frequent constituent of normal and quite commonly of pathological stools. It appears either as single cells or in pearly chains. It frequently shows budding. Usually the cells are oval, more rarely they are round. We find them both in firm and in diarrhœal evacuations, although they are more common in the latter variety. The reaction of the fæces seems to have no influence upon their growth, for I have met with them in alkaline as well as in acid stools. With Lugol's solution they are stained a yellowish or mahogany brown. Von Jaksch quite correctly calls attention to the occurrence in the fæces of bodies very similar to yeast cells, but I have noticed that they are for the most part smaller than yeast cells and have no double contour and do not bud. Furthermore, they differ from yeast cells in giving the same reaction as starch with Lugol's solution. They should very probably be classed with the clostridien (page 121).

(*c*) *Sarcina* (Goodsir) (Fig. 17).—Strange to say, the occurrence of genuine, fully developed sarcinæ in the fæces appears to be but very little known. They are not infrequently found in the stools of patients suffering from gastrectasia. Several times I have thus had my attention directed to an existing gastrectasia with masked symptoms. In one case, however, in which I found them in the evacuations there was no gastrectasia present. Usually the evacuations in which sarcinæ appeared in very large numbers were of a diarrhœal nature, and the question therefore suggests itself whether or not the migration of sarcinæ *en masse* into the intestinal canal can not give rise to fermentative processes in the latter. As already remarked, sarcina intestini in no wise differs from the sarcina ventriculi. As in the latter we also find, in addition to well-formed, brownish yellow or lighter coloured bundles, those small coccous forms whose constriction is indicated only by a loose cementing membrane. Like the sarcina ventriculi, the sarcina intestini also shows a distinct cellulose reaction with iodin and chlorid of zinc solution.

(*d*) *Bacteria.*—Undoubtedly bacteria form the greater percentage of the micro-organisms occurring in the intestines, or rather in the fæces. They consist both of micrococci and bacilli. According to von Jaksch, thin stools as a rule contain more bacilli and firm ones more micrococci. Some of these organisms are motile.

1. By far the most numerous are those bacilli first described by Escherich as belonging to the group of *bacterium coli*. They are quite thick, short rods, in part motile and partly immobile; in appearance and growth upon most nutritive media they very much resemble the typhoid bacilli. Unlike the typhoid bacilli, they do not curdle sterile milk and develop gases (CO_2 and H) in nutritive media containing sugar. Their size varies widely, ranging between 0.5 and 3μ.

Careful study of this variety of bacteria has shown that they are really made up of several groups resembling one another, and which, as it appears, can merge from one into the other, thus permitting the formation of pathogenic organisms from innocent saprophytes. Thus a large number of diseases have already been observed in which it is suspected that the bacterium coli plays an important rôle—e. g., cholecystitis and cholangitis, appendicitis, acute dysentery, peritonitis, etc.

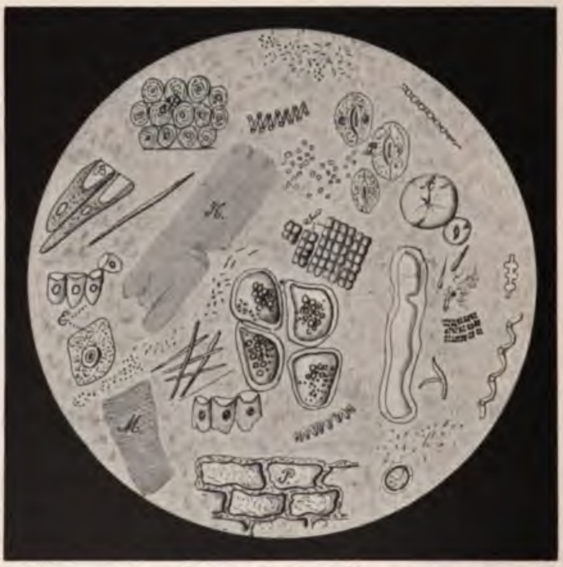

FIG. 17.—FÆCES FROM A CASE OF CHRONIC ENTERITIS, SHOWING SARCINÆ.

A, starch granule; *K*, fractured triple phosphate crystal; *M*, muscle fibres; *E*, epithelial cells; *B*, goblet cells; *P*, vegetable cells and fibres. (Original observation.)

The addition of one per cent of iodid of potash to the culture media, as advocated several years ago by Elsner,[39] but more particularly the well-known Widal's test,[40] enables us to distinguish typhoid bacilli from the colon and other bacilli.

2. *Bacillus Subtilis* (Hay Bacillus).—This bacillus, first observed in the fæces by Nothnagel, is very often met with under normal as well as pathological conditions. It forms either long, motile filaments with spores, or isolated bacilli with spores, or, finally, groups of spores. It is readily recognised by its thick contours as well as by its strongly refractive spores. It stains yellow or yellowish brown with Lugol's solution. It has no special significance.

3. *Bacterium Lactis Ærogenes.*—These are thick, short rods, 1–2μ in length and 0.5μ in width. They are immotile. They are characterized by their property of causing milk to curdle and to ferment with the formation of gas within sixty hours. On potatoes, also, the bacterium lactis forms gases. Besides milk sugar, they also cause cane sugar to ferment. It is possible that owing to its active gas-forming properties the bacterium lactis plays an important part in the etiology of meteorism; but of this we know nothing certain.

4. *Bacillus Putrificus Coli* (Bienstock).—These are slender rods about 3μ in length, which frequently form long threads. The spores are marginal, and develop at one or both ends; if at one end only, they give the bacillus a drumstick appearance. The bacillus, especially in the presence of air, causes a rapid decomposition of albumin with the formation of ammonia, amin bases, amido-fatty acids, tyrosin, phenol, indol, etc. I have very often observed the bacillus in stomach contents as well as in fæces, but, unlike Bienstock, would not consider it as a regular parasite of the fæces.

5. *Organisms that Stain Blue with Iodin.*—Nothnagel has observed a number of micro-organisms which stain blue with iodin, and he regards one variety as identical with the *clostridium butyricum* of Prazmowski. The bacillus butyricus (Fig. 18) forms rods from 3μ to 10μ in length and 1μ in width, and frequently occurs in chains. It is sometimes said to form long filaments which are quiescent sometimes, at other times mobile. Usually the bacillus butyricus is lemon- or lozenge-shaped and forms large clusters. It gives a very pronounced iodin reaction. The bacillus is anærobic. It causes an active fermentation of starches, dextrin, sugar, lactates, and cellulose. The bacillus butyricus is very probably the chief agent in the formation of butyric acid and of gases in the intestines. According to von Jaksch, with whom I fully agree, the bacillus butyricus is found in especially large numbers in pathological conditions of the intestines. In almost every normal stool we find it in smaller numbers. It does not have any pathological significance.

The *pathogenic* organisms occurring in the fæces include the cholera, typhoid, and tubercle bacilli. Since a work like the present one can not include a discussion of the acute infectious diseases, the *tubercle bacillus* is the only one we shall speak about.

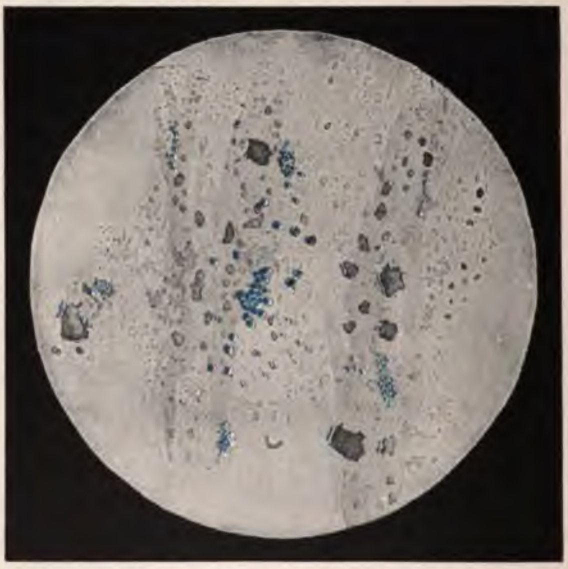

FIG. 18.—BACILLUS BUTYRICUS (*Clostridium butyricum*) STAINED WITH IODIN. (Original observation.)

Assuming its morphology to be well known, we would simply say regarding its demonstration, that the technic differs in no manner from that of sputum examinations, and it is entirely superfluous to enter here into the details of that procedure. As to the diagnostic significance of tubercle bacilli in the fæces, it may be summed up as follows: It is conclusive of a tubercular process in the intestines when we know positively that the material examined comes directly from the intestines—as, for example, in tubercular rectal ulceration. Otherwise the question of intestinal tuberculosis must remain in doubt.

The diagnosis, intestinal tuberculosis, can, however, be made with certainty where there is an entire absence of sputum, and tubercle bacilli constantly appear in stools that are of a thin, bloody, or purulent character.

Cocci are also found in the fæces, though not as frequently as bacilli. Since they are of much less practical importance, we shall refrain from their discussion, referring the reader to Mannaberg's able description in Nothnagel's work on Intestinal Diseases.

6. *Crystalline Bodies.*—These may be organic or inorganic, and are seen very often in the fæces. With the exception of the biliary concrements previously mentioned (see page 111), the diagnostic importance of crystalline bodies is very slight. Since they may be mistaken for other similar substances it is necessary to be able to recognise them. The following are the principal crystalline bodies which come into consideration:

(*a*) *Hæmatoidin Crystals.*—Von Jaksch has observed these in long-continued catarrhs following stasis, and also in many cases in which intestinal hæmorrhages had but a short time previously occurred. Usually hæmatoidin shows an indistinct crystalline structure; the crystals are in part free and in part embedded in a lustreless, mucoid mass. I have never been able to find the bodies mentioned by von Jaksch.[10]

(*b*) *Cholesterin.*—It is extremely rare to find cholesterin present as fully formed rhombic plates. This applies as well to normal as to pathological stools. Only once after the administration of nutritive enemata have I seen them in large quantities. Nothnagel has seen them after nutritive rectal enemata of peptone, wine, and eggs. This author quite correctly calls attention to the very deceptive similarity between fragments of triple phosphates and cholesterin scales. Indeed, the resemblance is so close that I have frequently only learned my error through obtaining a negative cholesterin reaction. Cholesterin also undoubtedly appears in normal fæces in an amorphous form; neither form has any diagnostic significance.

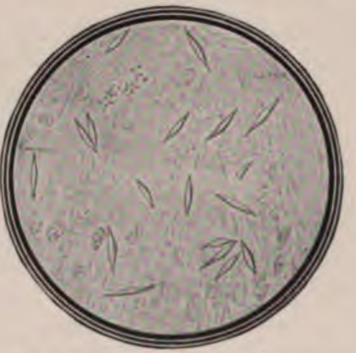

FIG. 19.—CHARCOT-LEYDEN CRYSTALS FROM FÆCES. (Original observation.)

(*c*) *Charcot-Leyden Crystals* (Fig. 19).—These are by no means extremely rare. They are identical with the crystals found in asthmatic sputum. In the same specimen they may vary very much in size. Sometimes they can be distinctly seen only with the highest power lenses. They are insoluble in alcohol, ether, and chloroform, but very soluble in hydrochloric acid and alkaline solutions. This permits of their ready distinction from fatty acid crystals. They stain with carmin, and can then be preserved for a long time. Nothnagel[5] has observed their appearance under most varied conditions—viz., reconvalescence from typhoid, typhoid at the height of the disease, phthisis, in dysenteric disturbances of the large intestines, in a rachitic child with firm stools, and in a child with profuse evacuations resulting from chronic catarrh of the large and small intestines. Perroncito[41] and Bäumler[42] first discovered the crystals in persons suffering from anchylostomal disease. This was the status of the question when Leichtenstern, in 1892, called attention to the coincident occurrence of these crystals with various intestinal parasites (ascaris, anchylostomum, trichocephalus, oxyuris, tænia, etc.), and stated that entozoa, whatever be their nature, were the most frequent cause of the formation of Charcot-Leyden crystals in the intestines, and of their subsequent appearance in the fæces. From this it follows, therefore, that the crystals possess great diagnostic value; if found in the fæces we may with the greatest probability, perhaps certainty, conclude that *the patient in question is suffering from intestinal entozoa of some kind* (Leichtenstern[44]).

Conversely their absence does not in any manner exclude entozoa. Leichtenstern based his views on the significance of the coincidence of the Charcot-Leyden crystals and entozoa, especially upon the fact that at autopsies he was always able to find them in those portions of the intestines in which, from experience, we know the parasites in question are usually found. In anchylostomiasis, in particular, these crystals are found with such constancy that he states that they are distinctly pathognomonic of that disease. Strange to say, little attention has been paid to this important discovery of so great a clinician as Leichtenstern, who has made excellent researches, especially regarding entozoa. The question has only been discussed in Roesen's dissertation[45], where, after an examination of seventeen cases of entozoa, an entirely negative result was reached. My own experience in the matter is too slight to demand recognition, but from the study of one case I have gained the impression that the value ascribed to them by Leichtenstern is correct.

The case in question was that of a gentleman thirty-six years old, who suffered from chronic catarrh of the large intestines, and whose stools I used frequently to examine very carefully. During one examination I found, in the mucous portions of the stool, Charcot-Leyden crystals both of moderate and very large size. Although not attaching much importance to my observation, I took note of it, and upon repeated examinations the same condition was observed. After three or four weeks the patient brought me tænia proglottids, whereupon a tænia mediocanellata was readily removed. In spite of most careful examination tapeworm eggs could never be found.

This case needs no further comment.* Leichtenstern's discovery is certainly worthy of greater attention than it has heretofore received.

(*d*) *Fatty Acids and Fatty Soap Crystals.*—We have already touched upon the most important points in this connection (pages 115 and 116). We here wish to add that the opinion expressed by Gerhardt[20] a long time ago, that the crystalline bodies occurring in acholic stools might be tyrosin crystals, has been wholly refuted by the investigations of Oesterlein[45], Stadelmann[46], Fr. Müller[15], and von Jaksch[10].

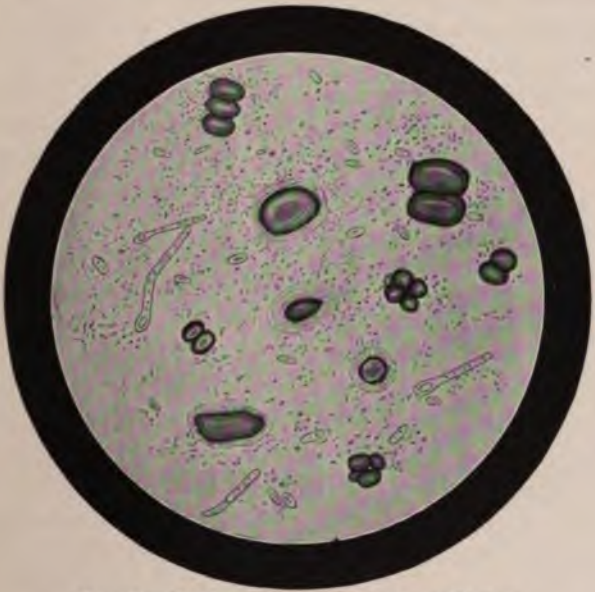

FIG. 20.—YELLOW CALCIUM SALTS FROM FÆCES.

In some the concentric arrangement of the layers is well seen. (Original observation.)

(*e*) *Phosphate of Calcium.*—This occurs in the fæces as neutral phosphate of calcium and as yellow calcium salts. The former consists of small or large wedge-shaped bodies, arranged for the most part in groups with converging apices. This variety does not absorb bile. Yellow calcium salts (Fig. 20) (whose acid has not yet been determined) usually assume irregular, oval, or circular forms. They are frequently fissured, and in some cases are arranged in concentric layers. The yellow colour comes from imbibition of bile. Neither form has any diagnostic significance.

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* It struck me as strange that when entozoa eggs were present the crystals were frequently missing. Besides numerous cases of tænia, ascaris, and oxyuris under my own observation, I would also mention a case of trichocephalus dispar in which Charcot-Leyden crystals were vainly looked for repeatedly by both my assistants and myself.

(*f*) *Oxalate of Calcium.*—This occurs in the well-known envelope form. The crystals may be small or large. They are found especially in patients under largely vegetable diet. These crystalline bodies are met with in normal as well as in pathological stools.

(*g*) *Ammonium-magnesium Phosphates* (Triple Phosphates).—These crystals are one of the most frequent crystalline elements of normal or pathological stools, especially fluid stools. They appear most frequently in the well-known coffin-cover shape, more rarely feather-shaped. The coffin-cover fragments frequently show tears and fractures (Fig. 17), and thus may occasionally resemble cholesterin crystals. The solubility of the triple-phosphate crystals in acetic acid enables us to avoid error in diagnosis. They have no diagnostic value.

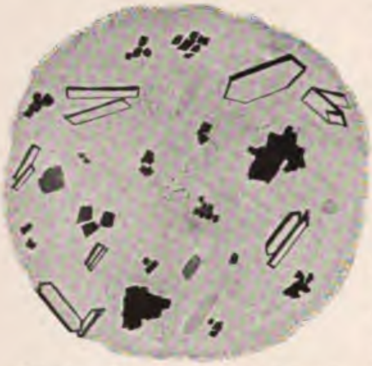

FIG. 21.—BISMUTH CRYSTALS FROM FÆCES. (Original observation.)

(*h*) *Sulphate of Calcium.*—The appearance of sulphate of calcium in the fæces is said to be extremely rare. I have never seen it in the dejections themselves, but once by the addition of sulphuric acid to fæces obtained some very pretty sulphate of calcium needles in the sediment, which consisted principally of bilirubin-calcium.

(*i*) *Bismuth Crystals.*—Very soon after administration of bismuth, irregular shaped, rhombic, dark brown or black crystals are excreted (Fig. 21). Until recently these have been regarded as sulphate of bismuth, but, as previously mentioned, Quincke showed that they consist of bismuth oxydyl.

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CHAPTER VI

THE DIAGNOSTIC VALUE OF THE EXAMINATION OF THE STOMACH CONTENTS IN INTESTINAL DISEASES

In the diagnosis of difficult intestinal diseases an analysis of the stomach contents is sometimes very important, or may even be decisive. In the first place, it enables us to exclude diseases of the stomach itself. Thus, for example, if we find a poorly defined tumour, from whose location alone it would be impossible to say whether it belongs to the stomach, the intestines, or the omentum, and where, in addition, there are general evidences of a serious diseased condition, the finding of normal stomach contents would in all probability speak against gastric cancer. On the other hand, abnormal stomach contents does not signify absence of the disease of the intestines. As an illustration of this by no means "rare complication," I may cite a very interesting case reported by Pulawski[1], in which the clinical picture was that of a dilatation of the stomach and a tumour in the epigastrium, while the autopsy revealed a primary cancer of the cæcum. The gastric tumour felt was occasioned by an aggregation of carcinomatous lymphatic glands which compressed the pylorus and caused a stenosis. The mucous membrane of the stomach was entirely normal. The reverse of this case is more frequently met with—i. e., a gastric tumour becomes displaced and simulates an intestinal neoplasm. Here, at times, examination of the gastric contents may clear up the diagnosis. What has always struck me as remarkable, and what also speaks for the diagnostic value of these examinations, is the fact that in cancer of the intestines, excepting where the tumour is very near to the pylorus, the gastric secretions may still be normal even in the most extreme stage of cachexia. Examination of the stomach contents may also give conclusive evidence for the diagnosis of a stenosis *below the duodenum.* It can, however, only indicate the existence of a stenosis, the nature of which must be determined by other means. In these cases the stomach contents are always bile tinged, especially in the fasting state. I was the first to show that, besides bile, pancreatic, and very probably, too, intestinal secretion, pass into the stomach. Hence we may sometimes observe the paradox, duodenal digestion occurring within the stomach. This can happen only where the gastric secretion, as is frequently the case, becomes weakened or entirely suppressed through the action of the intestinal fluids, especially of the bile. As has already been shown on page 44, when the gastric juice remains sufficiently acid no variations from the normal occur. We may even find an alternating reaction of the gastric contents, according as intestinal or gastric factors predominate. Cohn, Riegel, Hochhaus, Schule, Herz, and I, have published examples of this last-named condition.

The presence of pancreatic juice in the stomach can be readily demonstrated by its tryptic action. Its demonstration, I believe, might be of importance in determining the etiology of a stenosis, for with a palpable tumour in the region of the ascending portion of the duodenum, the occurrence of pancreatic ferments in the stomach contents would speak for, and the absence of such ferments against, the existence of a pancreatic tumour; still, I can not support this statement by actual cases.*

Besides the deeply situated duodenal stenoses, the much rarer ones of the jejunum can, I think, also be diagnosed through examining the gastric contents. In these cases the latter no longer contains pure bile, but a yellowish brown, slightly feculent material, which has the appearance and composition of jejunal chyme. Careful examinations of this, but more especially its constant appearance in the stomach contents, should give us a clew to the existing condition, particularly if we can exclude a fistula between the stomach and the small or large intestines. We would add that, unlike deeply seated duodenal stenoses, the suprapapillary variety can not be diagnosticated by examinations of the stomach contents, for the latter are exactly the same as in benign or malignant pyloric stenosis.

In some cases the determination of the acidity of the stomach contents may be important for the diagnosis of an intestinal affection—e. g., in the differential diagnosis of ulcer of the duodenum and of the stomach. Leube[2] states that he found normal acidity of the stomach contents in a case of duodenal ulcer, while, from the investigations of Jaworski and Riegel, we know that the acidity in gastric ulcer is very often increased. Too much value, however, must not be attributed to this symptom.

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* It is well worth repeating, although obvious to one well acquainted with the subject, that the finding of bile once, or even repeatedly, in the gastric contents possesses no diagnostic value.

Oppler[3] has lately and quite correctly directed attention to the total cessation of gastric secretion in chronic diarrhœas, a condition which has more of a practical than diagnostic value. As I can state from repeated personal experience in these cases, we may meet with severe forms of gastritis with entire absence of all the secretive factors (hydrochloric acid, pepsin, and rennet). I have no doubt that a distinct connection exists between the anacidity of the stomach and the chronic diarrhœa and intestinal catarrhs.

The condition of the gastric contents indirectly aids us in understanding certain intestinal disturbances, among others the chronic constipation that so often follows atony or ectasia of the stomach. In like manner, in displacements of the stomach, particularly when well marked, we can, as a rule, assume that a displacement of the intestine, especially of the large intestine, exists. The methods by which these intestinal displacements can be recognized have already been described.

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CHAPTER VII

THE DIAGNOSTIC VALUE OF URINARY EXAMINATIONS IN INTESTINAL DISEASE

THE examination of the urine is just as important in diseases of the intestines as it is in almost all other internal and many external diseases. It may in many cases substantiate and even clear up the diagnosis. Thus, to cite one instance, the discovery of an amyloid kidney may serve as a useful argument for the existence of amyloid disease of the intestines, a condition very difficult to diagnose. An attempt to dwell on all existing relations between the intestines and the urine would mean an account of almost the whole pathology of the urine. In the following recapitulation we confine our remarks solely to the clinical connection between intestinal diseases and certain abnormal substances found in the urine. These substances include:

1. *The products of intestinal putrefaction*, especially of albuminous putrefaction, the greater number of which, as we know, appear in the urine as aromatic combinations. These include indoxyl- and skatoxylsulphuric acids, as well as the ethereal sulphuric acids (skatoxyl, parakresol, and phenol ethereal sulphuric acids).

From the fundamental investigations of Jaffe[1], E. Salkowski[2], Baumann[3], Brieger[4], and Senator[5], we know that the indoxylsulphuric acid of the urine is regarded as a derivative of the indol formed within the intestinal canal. In the intestines indol is already oxidized to indoxyl, and the latter combines with the sulphuric acid, or with the sulphates derived from the food. Indoxylsulphuric acid (indican) appears in the urine in the form of the last-mentioned combination. From this it follows that indican is an entirely normal product of intestinal metabolism, and that only its increase is to be considered as pathological. Whereas the normal daily excretion of indican varies from 5 to 20 milligrammes (Jaffe), in increased intestinal putrefaction it may reach 100 to 150 milligrammes. Fr. Müller[6] and Ortweiler[7] very correctly consider as necessary for increase in the urinary excretion of indican a certain percentage of albumin within the intestines, marked stagnation of the intestinal contents, and finally a certain absorptive faculty on the part of the bowel. According to the animal experiments of Jaffe (which have been fully verified in man), increased excretion of indican occurs mainly in obstructions of the small, and to a less extent of the large, intestines. Nevertheless, we may also have an increased indican excretion in stenoses of the large intestines associated with long-continued putrefaction of their contents. Should suppuration coexist, the indicanuria will equal in amount that of the small intestines.

As may be readily seen, increased indicanuria is largely dependent upon intestinal peristalsis; other things being equal, the slower the peristalsis the greater will be the amount of indican in the urine, and *vice versa.* The more putrefactive products carried off by the intestines, the less will be brought to the kidneys. But this rule holds good only in a general sense: for Jaffe, Vries, Ortweiler, and von Pfungen (with whom Nothnagel agrees) have shown that there may be no indicanuria in obstinate constipation of the worst type. The reason very probably is that, in consequence of increased desiccation of the fæces, few putrefactive products are absorbed, and the fæces therefore very rapidly undergo further decomposition.

The above considerations indicate that the presence of indican in the urine is of decisive value only when taken in connection with all other clinical symptoms. Indicanuria may be of importance in the differential diagnosis between stenoses of the large and small intestines, or between benign and malignant pyloric stenosis, etc. The almost constant increase of indican in suppurative peritonitis is also important.

Besides indigo blue there is a second urinary colouring substance, indigo red ("indigrubin," Rosin[8]). Rosenbach's well-known reaction depends upon its presence. As shown by control tests of E. Salkowski[9], C. A. Ewald[10], Abraham[11], Rumpel and Messter[12], and others, Rosenbach's reaction, like a pronounced indigo-blue reaction, indicates nothing more than an increase in the excretion of indigo—i. e., albuminoid putrefaction. To a certain degree, the ethereal sulphuric acids of the urine are indicative of putrefaction. As with indoxyl, it has been shown that albuminoid putrefaction of stagnating intestinal contents is also an important factor in the production of the etherial sulphuric acids (E. Salkowski, C. A. Ewald, Baumann, Kast and Baas). A second undoubted factor is disturbances in function, such as occur in ileus, incarceration of the small intestines, lead colic, peritonitis, intestinal tuberculosis, and cholera. Finally, Brieger's[13] investigations have shown that in such acute infectious diseases as diphtheria, scarlet fever, and facial erysipelas there is an increase of ethereal sulphuric acids, while in typhoid, recurrent and intermittent fevers, variola, and meningitis there is a decrease.

Increase in the ethereal sulphuric acids has also been observed in foul-smelling and putrid processes in various portions of the body. Furthermore, we may mention that even in healthy persons the amount of ethereal sulphuric acids excreted is subject to great fluctuations, and also that very much depends upon the nature of the food ingested. It therefore follows that the excretion of the ethereal sulphuric acids occurs under various conditions; that it depends as much upon their formation within the intestines as it does upon their absorption; furthermore, that it is to a large extent dependent upon the diet, and that even normally it is subject to the greatest variations. In the present status of the subject it would be very risky to form any positive diagnostic conclusion from an increased excretion of the ethereal sulphuric acids. Only where there is a marked *abnormal* increase can any conclusion be drawn, and then only in conjunction with other signs.

2. *Acetonuria and Diaceturia.*—Petters[14] and Kaulich[15] pointed out the connection between digestive disorders and acetonuria. Later, Litten[16] described the occurrence of acetonuria in dyspeptic conditions. Acetonuria in diseases of the digestive tract has been most carefully studied by Lorenz[17]. He found this condition present in diseases of the stomach, as well as in the greatest variety of intestinal disorders—e. g., gastro-duodenal catarrh, gastro-enteritis, intestinal obstruction (from marked coprostasis), tænia, peritonitis, and perityphlitis. As fever was present in the last-named conditions, febrile acetonuria could not be positively excluded. In almost all these cases, in addition to the aceton, diacetic acid was also found. At present no diagnostic significance can be attached to acetonuria.

3. *Albuminuria.*—Maixner[18] and Pacanowski[19] first called attention to the occurrence of an enteric peptonuria (more correctly albuminosuria), and Robitschek[20], who employed improved methods, confirmed their discovery; nevertheless, the doctrine of an enteric albuminuria rests upon a very weak basis. Chvostek and Stromayr's[21] recently published investigations upon the occurrence of

alimentary albuminosuria in ulcerative intestinal tuberculosis are deserving of greater attention. A few hours after such patients had received large amounts of dry peptones or of somatose, albumoses were found in the urine, Devato's[22] and Salkowski's[23] methods being employed for their detection. In some positive cases of ulcerative intestinal tuberculosis albumoses were not found, so that a diagnostic value can only be attached to a positive result of the test.

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GENERAL THERAPEUTICS OF INTESTINAL DISEASES

"*Non medicamentis confidere sed therapiæ.*"—VON LIEBERMEISTER.

CHAPTER VIII

THE DIETETIC TREATMENT OF INTESTINAL DISEASES

THE fundamental principles laid down in the general section of my book on The Diagnosis and Treatment of Diseases of the Stomach apply equally to the general dietetic treatment of intestinal diseases. To avoid repetition, we refer the reader to that work. Several of the rules of procedure there described—e. g., those for the treatment of gastric ulcer and acute gastritis—may with slight changes be applied to cases of ulcer of the small intestine and acute enteritis. The conditions are, however, far more complicated in many other intestinal diseases. According to the pathological condition in the intestines, the stomach contents must have a varying influence upon the intestinal contents; these latter are acted upon by three distinct secretions, each of which may vary in its effect upon the intestines. Absorption from the intestines and intestinal peristalsis may vary considerably. Some excrementitious substances from the blood exert an undoubted action on the intestines. Nervous and vaso-motor influences can hasten or retard digestion. From a consideration of all these conditions it is evident that intestinal digestion is a very complicated process. The principles of feeding in gastric affections are based to some extent on the results gained from an analysis of the stomach contents which can be readily obtained. On the other hand, we have as yet no satisfactory and practical general method of obtaining and analyzing the intestinal contents. Sometimes an idea of the changes which the food has undergone in the bowel may be gained from an analysis of the fæces. Hence the pathologist and physiologist have for many years paid great attention to the examination of the fæces.

The first fundamental fact was discovered by Rubner, who showed that the digestion of food stuffs depends principally upon their chemical and physical composition.

The following table is taken from Rubner's[1] work:

FOOD STUFFS.	WEIGHT OF SAME IN GRAMS.		ABSORBED IN PER CENTS OF				
	Fresh.	Dried.	Dried substance.	Albumin.	Fat.	Carbo-hydrates.	Ash.
Meat	884	376	95	97	95	. . .	82
Eggs	984	247	95	97	95	. . .	82
Milk	2,470	315	92	94–99	95–97	100	51
Milk and cheese	2,490	420	94	96	97	100	74
White bread	860	753	95	81	. .	99	93
Black bread	1,360	765	85	68	. .	89	64
Macaroni	695	626	96	83	94	99	76
Indian corn	750	641	93	85	83	97	70
Corn and cheese	. . .	780	96	93	91	96	81
Rice	638	552	96	80	93	99	85
Peas	600	521	91	83	. .	96	68
Potatoes	3,078	819	91	68	96	92	84
Cabbage	3,830	406	85	82	94	85	81
Carrots	2,566	352	79	61	94	82	76

This table is very instructive in that it shows that the albuminous substances contained in meat, eggs, and milk are almost totally used up in the intestines. None of the vegetable matters, with the exception of Indian corn, are so completely absorbed. Very little of black bread, potatoes, and carrots is absorbed. The greater part of the carbohydrate materials is, however, absorbed in the intestines. Thus milk loses 100 per cent; white bread, macaroni, Indian corn, rice, and peas almost as much; potatoes and carrots lose little of their carbohydrate matters in the intestinal canal. Intestinal absorption of the fatty matters is also very active; thus milk loses very much of its fat, as do also meat, eggs, macaroni, rice, potatoes, cabbage, and carrots.

The digestibility of food stuffs can be further heightened by the manner of preparing them. Thus, according to Rubner, Boeck, Praussnitz, Weiske, and others, 72 per cent of the albuminous matters are absorbed in the intestine from boiled peas as against 82 per cent from ground-up peas, and 68 per cent of boiled potatoes as against 80 per cent of mashed boiled potatoes. The addition of spices to the food is also of undoubted benefit in this respect. The above-mentioned figures apply only to healthy persons. Unfortunately, our knowledge of the digestibility of food stuffs in diseased individuals is very limited. The investigations of Fr. Müller[2], so frequently referred to, have shown that in intestinal diseases the absorption of fats is early disturbed, and the absorption of nitrogenous matters is interfered with only in some intestinal catarrhs.

The absorption of carbohydrates is almost never interfered with, although it is probable that some of the carbohydrates are lost through fermentative processes in the intestines.

The food stuffs exert an important influence upon many of the intestinal putrefactive processes. Thus Rubner[1] observed that after a diet of bread the products of albuminoid putrefaction disappeared from the urine, and in their stead there appeared a butyric acid fermentation.

Hirschler[3] demonstrated that a direct antagonism existed between albumin and carbohydrate putrefaction. Ortweiler[4] showed that a free diet of carbohydrates is apt to be followed by the complete disappearance of indican from the urine. It is evident that the food stuffs vary in their proneness to decomposition, and that in disease there may be a greater or less tendency for the occurrence of putrefactive processes of the albuminous, carbohydrate, or fatty matters. Unfortunately, our knowledge of the principles of dietetics is so meagre that no deductions can be drawn from this.

In the absorption of food stuffs from the diseased intestine another factor is of importance, viz., the influence exerted by the food upon the intestinal functions. Thus, for example, in some diseases of the intestines milk causes constipation, in others diarrhœa. Eggs, to take another example, are not well borne in diarrhœa, but are always prone to cause flatulence. The idea that some substances are easier digestible and others not so is gradually losing many adherents, for we are gradually coming to the conclusion that the digestibility of foods varies in each individual case. This illustrates the uselessness of basing our dietetic measures solely upon the digestibility of various foods. Clinical experience must teach us the principles of dietetics. From my observations and those of others the following rules may be laid down:

1. In a number of intestinal diseases a change of diet is unnecessary, or may even be harmful.
2. In some cases special dietetic restrictions are directly indicated, but these should be as few as possible.
3. In another series of cases an abundant heavy, not easily digestible or absorbable diet is indicated.
4. The general aim of our treatment should always be to so manage the case before us that digestion of a normal diet will always occur in the alimentary canal without any subjective or objective disturbances. Under these circumstances only can the case be considered cured.

The reasons which have led me to adopt the above principles of treatment will be given in what follows, which will contain a general description of the principles of feeding in diseases of the intestines. For simplicity of understanding, intestinal diseases are classified * as

1. Diseases of the mucous membrane (ulcers of the duodenum, jejunum and ileum, acute and chronic enteritis, carcinoma, intestinal ulcers in general).
2. Functional disturbances (increased or diminished peristalsis).
3. Stenosis or occlusions.
4. Localized peritonitis (appendicitis); diffused peritonitis.
5. Neuroses.

1. Diseases of the Intestinal Mucous Membrane

In diseases of the mucous membrane the principles of feeding are similar to those laid down for the treatment of affections of the body in general—i. e., rest for the affected part. The temporary withdrawal of all foods constitutes the greatest possible amount of rest for the digestive tract, and one that is often followed by brilliant therapeutic results. This method comes up for a special consideration in acute bleeding from the stomach or intestines, in acute gastro-enteritis, and in rebellious forms of gastric or duodenal ulceration. The importance of the latter disease is worthy of special consideration in this place. That the *absolute* withdrawal of food is the best therapeutic measure in acute intestinal hæmorrhage (exclusive of bleeding from hæmorrhoids) is not as yet, according to my experience, generally acknowledged. I emphasize the word absolute, because I believe that in these cases even water by the mouth is harmful. In such a patient the physician, and especially the relatives and friends, are but too apt to give the patient large quantities of fluid to drink to compensate for what has been lost by the bleeding—a procedure which I consider absolutely wrong. Food should not be given by the mouth until twenty-four to thirty-six hours after the hæmorrhage has ceased. By that time the bleeding vessels have probably been closed by a thrombus. If the weakness of the patient calls for immediate feeding, nutrient enemata should be ordered.* During the following few days the patient should be kept on a fluid diet. After eight to ten days the diet may be gradually increased according to the well-known plan of Leube.

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* In this classification it may occur that some diseases might, during their course, come under another heading than they would at first; thus, a carcinoma of the intestines might be at first considered a disease of the mucous membrane, later, as the symptoms of obstruction come to the foreground, as a stenosis. The same is true of many other intestinal affections.

In acute hæmorrhage from the large intestines (as in hæmorrhoids, dysenteric ulcers, and neoplasms) the danger of profuse bleeding is not great, and hence the entire withdrawal of food is unnecessary. It will generally suffice to restrict the patient to a soup diet for a few days.

In chronic bleedings, which are apt to be small ones, it is very important so to arrange the patient's diet as to prevent the symptoms of exhaustion which are apt to appear. The choice will largely depend upon the nature and character of the illness. In the special part of the present work more attention will be paid to this subject.

In violent hæmorrhages from large blood-vessels (embolism of the mesenteric artery, rupture of varicose veins, etc.) we are usually powerless. Diet has but little influence upon these hæmorrhages.

The treatment of acute gastro-enteritis or enteritis alone is far simpler. In these affections the loss of appetite facilitates the withdrawal of food. In very acute diarrhœas we must provide against the severe thirst which is so apt to be present, by giving the patient diluted claret, oatmeal soups, cocoa boiled in water, and tea without sugar. In chronic duodenal ulceration the same diet as in gastric ulcer is to be ordered. The patient should remain in bed for several weeks, poultices should be applied to the abdomen, and he should partake of a bread-and-milk diet. This diet should be continued until subjective and objective sensitiveness have disappeared, when the patient should abstain for a long time from injurious diet—that is, food digested with difficulty (ice cream and iced dishes, raw fruit, hard bread, all kinds of cabbage)—and improper drinks (iced drinks, acid fruit juices, carbonated beverages).

In very obstinate forms of duodenal or jejunal ulceration the treatment is similar to that of gastric ulceration—i. e., abstinence from all food for ten to fourteen days, and rectal alimentation. In nine undoubted cases of this class I found that the above-outlined treatment was always sufficient.

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* Concerning the composition and method of giving nutrient enemata, see Boas, Diagnosis and Treatment of Diseases of the Stomach, fourth edition, Part I, p. 293.

The diet in chronic enteritis varies greatly, according to the situation, intensity, extent, and duration of the disease. It is difficult, therefore, to lay down any general rules, and we will limit ourselves to a few suggestions, reserving fuller details for the second part of this work. The treatment will depend mainly upon our being able to locate the intestinal catarrh.

Catarrhal affections of the small intestines call for a particularly cautious diet. In long-standing diseases it is clear that the intestinal secretions are deficient in quality, and hence can not act in a normal manner upon the chyme. It can be readily understood that if, in place of the normal strongly alkaline intestinal juice, a weakly alkaline one containing large quantities of mucus is secreted (in which cases the acidity of the chyme is not sufficiently neutralized), the pancreatic juice will be acted on in an abnormal manner. In the presence of the strong acidity the pancreatic juice loses its tryptic, diastatic, and fat-splitting properties; the emulsification of fats is delayed, or does not at all take place; the action of the bile is likewise interfered with by the hyperacidity of the small intestines. Where, as is usually the case in intestinal catarrh, there is diarrhœa, the greatest care must be exercised in the selection of a suitable diet. If the diet does not contain sufficient nourishment, the patient may suffer from a transitory loss of albumin. Nevertheless, this plan must be carried out. The more numerous the evacuations, the greater is the need for restriction of the diet. In the beginning, gruel, cocoa, tea, beef tea, nutrose,* and claret, or cognac diluted with water, may be ordered for the patient. The diet must be varied according to the number and consistency of the movements. Regarding a diet of milk and eggs in diarrhœa, the most important facts have already been given; as the patient improves, an egg may be stirred into the soup, and according to the manner in which the patient stands this the diet can be still further increased. We may then gradually let the patient take milk. Its effects should be carefully noted; very often it will have to be entirely excluded. It is obvious that all substances that increase peristaltic motion (fruit, cider, certain wines, [organic] acids, honey, sugar) must be avoided. It is not correct, however, as is still often done, to give general instructions according to the above-mentioned principles. Exact instructions as to the diet must be given in treating each individual case.

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* In all diarrhœal conditions somatose must be avoided, because it is apt to aggravate them.

In catarrh of the large intestine dietetic principles are quite different. There is present either constipation, or constipation alternating with diarrhœa, or diarrhœa alone. In the last-named variety the diet is the same as that of catarrh of the small intestines. In the other forms our aim should be to regulate the movements without injury to the inflamed intestinal mucous membrane. In these cases digestion in the stomach and the small intestines is usually normal, and therefore the patient may from the very beginning be placed upon a full and nourishing diet. We may begin with a free diet (meat, fish well prepared, soft-boiled eggs, fat such as butter, chicken fat, cocoa butter). Vegetables in *purée* form and carefully prepared pastry may also be allowed. Conversely, experience has shown that soups tend to arrest peristalsis; they, as well as other physiological astringents (rice, farina, cocoa, red wine, huckleberry wine, etc.), are to be avoided. Physiological laxatives (honey, milk, sugar, glucose, grapes, stewed fruits, and sweet pastry) are especially to be recommended. In these cases milk, and particularly such of its derivatives as contain acids (buttermilk, koumyss, sour milk), may be employed to great advantage. We should strictly forbid the use of all substances which from experience resist all action of the intestinal secretions, such as the cellulose of cabbage, beets, and root vegetables, as well as indigestible seeds which are contained in many fruits (huckleberries, cranberries, currants, and gooseberries). A very similar diet is to be ordered in that form of intestinal catarrh that manifests itself in diarrhœa alternating with constipation, for here the constipation must be looked upon as the predominant and primary factor. In these cases our judgment of the results of the treatment instituted should not be governed by the effect upon the constipation alone, but from the appearance of the evacuations, and particularly from the amount of mucus contained in them. At the same time the above-named general principles must not be lost sight of. In primary catarrhs of the lowermost portion of the intestine (sigmoid flexure and rectum) the same general principles also apply.

In *secondary* catarrh, we may under favourable circumstances achieve successful results by removing the cause of the congestion in the intestinal blood-vessels. Generally we will have to content ourselves with a symptomatic treatment of the intestinal disturbances, following the above-mentioned underlying principles.

The dietetic treatment of *intestinal* ulcers also varies much, according to their nature and situation and the influence which they exercise upon the movements. Thus, according to Nothnagel, ulcers of the small intestine or of the ascending colon only cause diarrhœa when they occur simultaneously with other conditions (catarrh, amyloid disease, etc.), while ulcers of the lower division of the large intestine are usually accompanied by diarrhœa. Naturally the diet in ulceration of the upper portions of the small intestines will have to be a very cautious but highly nutritive one, and in ulcers of the lower portions of the small intestine we should by all means avoid all irritation of the mucous membrane from indigestible food masses. For the rest, the condition of the evacuations (consistency, number, admixtures such as pus, blood, etc.) will have to guide us in the choice of the diet.

As regards *tumours*, the malignant ones alone require special dietetic measures. We will have to take into consideration the preservation of the patient's life, and, if possible, the improvement of his general condition, the local (intestinal) damage (secondary catarrh, stenosis). Since our dietetic measures will ultimately depend upon these complications, they will, to avoid repetition, be detailed in the section on "Intestinal Stenosis" (Part II).

2. Diet in Functional Disturbances of the Intestines (*Retardation or Acceleration of Peristalsis*)

Under this heading, in the first place, we include habitual constipation and habitual diarrhœa. Both conditions may be accompanying symptoms of other diseases, or they may occur as independent functional intestinal disturbances; they therefore deserve to be discussed separately. In the second portion of this work further consideration will be paid to this subject; in the present place we wish to discuss the dietetic treatment of simple, uncomplicated constipation and diarrhœa.

In habitual constipation the diet must be a plentiful as well as a mixed one, and should contain substances that are known to stimulate peristalsis. Conversely, as already stated, dietetic astringents must be avoided. The first indication is frequently disregarded. We have already seen that foodstuffs are divisible into those well utilized and those poorly utilized by the economy. The first group includes all fluids and substances dissolved in them, meat, fish, wheaten bread and the like, light pastry, etc. The second group includes those foodstuffs that leave more or less of an indigestible residue—as, for example, fruit, rye bread, cabbage, potatoes, salad, and other vegetables rich in cellulose.

The main feature in the treatment of habitual constipation is the employment of this last-named variety of foodstuffs. Were a vegetable diet as nutritious as a mixed diet, and were its exclusive employment not frequently associated with objective (gastric and intestinal dilatation) and subjective disturbances (abnormal flatulence), it would be the ideal one in constipation, since it leaves behind a considerable indigestible residue. In general, therefore, sufferers from constipation should be given a mixed diet containing a minimum amount of totally digestible foodstuffs and a preponderance of those that contain much indigestible material. With such a *régime* we may accomplish much, but not all we wish to. The so-called "physiological laxatives" must also be given. We know a number of them; they act either chemically or thermically. The *chemical* ones include the organic acids, the mineral salts, and numerous sugars as well as fats. The organic acids include lactic, butyric, acetic, and probably also a number of other acids developed in the body from carbohydrates (formic, caproic, caprylic, propionic acids, etc.). As the investigations of A. Bokai[5] have shown, these acids are important factors in peristaltic action. It is a question whether the acids introduced as such, or those which develop from fermentation in the intestines, are the main causes of normal peristalsis; for many reasons the latter variety should be regarded as the main cause. It is certain, however, that these acids do excite intestinal peristalsis very strongly—more strongly, for example, than do many vegetable cathartics.

Among the foods which contain these acids are buttermilk, sour milk, kefir, and koumyss. In the latter two substances the carbonic-acid gas is also an efficient factor, for this gas excites intestinal peristalsis.

We know from animal experiments and observations upon man that the mineral salts also stimulate peristalsis. Upon this fact are based our ideas of the action of the muriated and Glauber salts. This has taught us to give patients suffering from habitual constipation plentifully salted food, such as sardelles, herring, caviar, smoked and boiled ham, and smoked beef.

The sugars have long been known as home remedies in habitual constipation. The sugars have not, however, a common action on the organism. Thus milk sugar, and probably also levulose and dextrose, have a greater influence upon peristalsis than has cane sugar. The physiological action of the sugars may be due to either the products of the decomposition of sugar in the body—i. e., lactic acid, butyric acid, acetic acid, etc.—or the sugars may exert an influence upon the intestinal mucous membrane by causing an active transudation from it. The investigations of Strauss[6] have shown that after the ingestion of sugar considerable transudation from the gastric mucous membrane occurs—a condition which I found present years ago in experiments on the gastric digestion of sugars.

The sugars have, therefore, a similar action to the saline cathartics (magnesium sulphate, sodium sulphate). Any one of a large variety of saccharin foodstuffs can be made use of instead of sugar itself. Among these may be mentioned sweet fruits, grapes, honey, sweet milk (also that sweetened with milk sugar), condensed milk, sweet wines (Tokay, Marsala, Sauterne, pasteurized grape juice, young wine, etc.).

Finally, the *thermic methods* should be mentioned. It is a fact of daily observation that cold applied externally or internally stimulates peristaltic action. Many of the laity, being cognizant of this result, are accustomed to drink cold water on an empty stomach in order to stimulate the activity of the bowels. It is advisable, therefore, in these cases to order cold instead of warm or hot, and preferably fruit soups, cold tea, coffee, sweetened lemonade, etc.

By means of these substances we are able to increase the intestinal activity at will. In rebellious cases of constipation one must not, as is so often done, expect too much from one particular remedy. Since these agents are adjuncts one to the other they must often be combined. I am accustomed to name a diet of this kind a "constipation diet," and I shall speak of it more in detail in the second part of this work. In the selection of appropriate food, the location of the trouble, its duration, and the condition of the patient have to be considered, but the principles upon which this diet of constipation is based must never be lost sight of, and always be applied in the proper manner.

As already stated (page 145), it is of prime importance to
lude all physiologically constipating remedies from the diet.
s I have often seen patients for whom the diet had been pre-
ed correctly in all respects, except that they were allowed to
ake of one half or even one bottle of claret daily—an absurd
wance. For similar reasons, cocoa, rice, farina, and foods pre-
ed from flour, unless freely sweetened with sugar, should be
rbidden. I have little doubt that a diet based on principles as
bove described and varied for each individual case, will result in
anent cure of even rebellious and long-standing cases of

constipation—a cure which will satisfy the conditions already mentioned (page 141), that even with ordinary diet the bowels will move in a normal manner. In those cases where the patients have been dosed with cathartics many years, only a carefully prescribed diet, strictly followed out for years, may result in a cure.

Besides the regulation of the diet, other therapeutic measures have to be observed in habitual constipation. Of equal importance are the regulation of the amount of exercise, stated hours for meals, a regular and sufficiently long time for defecation, a stimulation of the weakness of the intestinal muscle by exercise of the muscles in general, in particular those of the abdominal wall, etc. These considerations belong rather to the special treatment of habitual constipation, and will be discussed in detail in the second part of this work.

The choice of diet in *chronic diarrhœa* depends, as has been mentioned above (page 144), upon the number and consistency of movements and the duration of the affection. The most important principles of treatment have already been considered in speaking of catarrh of the small intestine. The mechanical, chemical, and thermal agents which stimulate peristaltic action in habitual constipation have, on the other hand, an unfavourable influence on diarrhœa. In regard to the first agent mentioned, we would therefore carefully avoid all coarse, indigestible, or not easily digested substances, and particularly those containing much cellulose, such as cabbage, salad, pickles, beets, and other root vegetables. Of the chemical agents, all organic acids and sugar are to be avoided. Where the food or drink has to be sweetened, it is best to use saccharin—a substance which Gans[7], in his investigations made in my laboratory many years ago, showed was also to a certain degree an intestinal antiseptic. *That good fats even in large quantities are generally well borne in chronic diarrhœa, is worthy of mention. Experience has taught me that even in severe cases one can prevent exhaustion by adding fats to the food.* All cold drinks, ice water, seltzer water, and other carbonized beverages (beer, champagne, etc.), and even cold water, should be avoided. Hot drinks, on the contrary (tea, soups, and claret), have a tendency to diminish peristalsis. As has been mentioned on page 144, milk is not, as a rule, well borne, and is apt to increase the number of stools. Cold milk has a very unfavourable influence on diarrhœa, while hot milk may not be harmful. Sometimes milk even in large quantities is well borne when cognac, limewater, powdered lime, talcum, ground barley, rice, Indian meal, or oatmeal, etc., are added to it. In a very few but convincing cases I have seen good results from systematic milk cures, and I believe that a careful trial may be made of them. It is hardly necessary to add that in these cases salts (table salt, nitre, and other spices) should be forbidden. If eggs (even poached) or foods containing them are not well borne they should be excluded from the diet. Generally, at least in the beginning of the treatment, eggs should be sparingly or not at all given.

In addition, those kinds of foods or drinks should be recommended which *from experience*, or from whose composition, we know tend to lessen peristaltic activity. In this category we may mention the cereals which contain much *mucin* (farina, rice, barley, oatmeal, porridge, etc.). These may be ordered in the form of soups, or, better, as thick, unsweetened gruels.* Other articles of diet which should be mentioned here are those which are characterized by containing more or less tannic acid. Among these may be mentioned "eichel cacao," "eichel coffee," infusion of nut leaves (e. g., butternut), of bearberry leaves (folia uva ursi), decoctions and jellies of fresh huckleberries, and a number of red wines containing much tannin, of which huckleberry wine is most useful.

A good old Bordeaux wine, the Greek wines (particularly Camarete), the Italian red wines, and Simaruba wine may be also given.

If the patient will adhere strictly to this diet not only during the whole course of treatment, but even, though not so strictly, for many months thereafter, his intestinal disturbances may be much improved or entirely cured. In modern society, with its dissipation and excesses, this plan of treatment will be adhered to only by a small proportion of the patients. Diet, however, is not all-sufficient. It must be remembered that other conditions (cold, constitutional disturbances, sexual excesses, overexertion) may have considerable influence upon the affection. This will be treated of more in detail in Part II of this book.

3. THE DIET IN CHRONIC STENOSIS AND OBSTRUCTION OF THE INTESTINES

Chronic stenosis occurs in different portions of the intestine, either as a benign (adhesive peritonitis, contracting scars of ulcers, benign tumours) or as a malignant process (carcinoma, sarcoma). No matter where the location of the disease and how great its severity, the main therapeutic object must consist in the removal of the causal factor. Diet will naturally have some influence upon this. Even if its location be recognised, we are often able to remove the cause of the obstruction only by operative means; still, the proper regulation of the diet may contribute much toward ameliorating or removing subjective or objective symptoms. In such cases two points have to be considered before beginning the feeding: the location and the degree of the obstruction. The location of the disease may be discovered in many if not all of the cases. The severity of the symptoms, the shape, consistency of the stools, the visible or palpable peristaltic restlessness and intestinal rigidity (Nothnagel), the appearance of meteorism, vomiting, etc.—all these signs and symptoms indicate, though sometimes falsely, the amount of obstruction. Since there are always processes of putrefaction above the stenosed portion of the intestine, the direct bearing of the amount of obstruction upon the diet to be prescribed is not to be underestimated. The diet will differ according to situation of the obstruction in the large or in the small intestine. The chyme remains fluid or semifluid until it reaches the lowest part of the ileum, and hence can pass through a greater obstruction here than below this point. Abnormal processes of fermentation and putrefaction are less apt to occur. The diet should be so arranged, therefore, that only fluids or foods prepared in liquid form are taken, while solid, indigestible, or not easily soluble matters are to be avoided as far as possible. It is hardly necessary to point out that food that ferments easily, or is already in a state of fermentation, should be excluded from the diet.

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* In these cases saccharin may be used with much advantage instead of sugar.

When the obstruction lies in the large intestine the conditions are far more complicated. In this part of the bowel the fæces lose most of their water, and in conditions of obstruction this loss of water is further increased because of the lack of fluid in the surrounding tissues. It matters not how large an amount of fluid the patient imbibes, the fæces are always firm when they reach the site of obstruction. One often has to ask himself the question whether it might not be advisable to order solid instead of liquid food. Under a solid diet, however, there might be danger of increased fermentation and putrefaction because of the large quantities of easily putrefactive material which would reach the site of obstruction, and furthermore, on account of the spasmodic peristalsis, solid indigestible substances might become lodged in the stenosed portion of the bowel. Lentils, peas, beans, asparagus, fruit, etc., in short, those vegetables which contain much cellulose, would be most apt to create such a condition—one very similar to that which occurs in a typical manner in stenosis of the pylorus. A number of cases have been reported in which moderate errors of diet have caused a decided increase or an acute exacerbation of mild or quiescent symptoms.

The diet in stenosis of the large intestines will therefore have to be based on the following principles: 1. It should contain a plentiful amount of solid material. 2. These solid materials must be free from mechanically irritating ingredients. 3. The diet must contain laxative ingredients. 4. These must not be excessively prone to undergo fermentation. The first two points need no explanation; some attention must, however, be paid to the last two. That substances which tend to make the stools less solid are advantageously added to the diet has been already clearly shown (page 147). Unfortunately, however, some of these substances, and among them the sugars, form a good substratum for the occurrence of fermentative processes above the site of the obstruction; hence they must be avoided. Experience has taught me that organic fatty acids and the acids of fruits are generally well borne. The same is true of salts, which probably diminish and certainly do not increase fermentation in the intestinal canal. A plan of diet based on such principles may be given in short *résumé* in the following: Mixed food—meat, fish, vegetables, etc.—should be prepared in *purée* form; well-salted and pickled meat or fish are especially recommended. Fat may be given in moderate quantities. Sweet milk is not well borne, but buttermilk, thickened milk, kefir (free from carbonic acid and two days old), koumyss (free from carbonic acid) are permissible. No, or only small quantities of, sweet stewed fruit, in which saccharin may be used instead of sugar, should be allowed. A diet suitable for each individual case may be constructed on these principles. It is of prime importance to counteract the tendency to severe grades of constipation; and if, as is often the case, the regulation of the diet will not suffice, one must occasionally order mild vegetable laxatives. It would be a palpable error to order a constipating diet for—or, what is still worse, to give opiates to—a patient who has partial intestinal obstruction. An exception to this statement is furnished by those cases of spasmodic tetanic intestinal peristalsis, in which opium not only quiets the contractions, but even acts as a laxative. This subject will, however, be treated of in another place.

The diet to be used in complete intestinal obstruction differs essentially from that in partial obstruction. In complete obstruction the diet must be very limited. Obviously this is of paramount importance when the obstruction is in the small intestines, for in these cases the entry of food into the stomach or intestines causes immediate vomiting. When the obstruction is in the large intestines (invagination, volvulus, foreign bodies, fæcal impaction) this is also, though in a less degree, true. Only small quantities of very easily digestible foods, preferably fluids, should be given at long intervals, and, when possible, nourishment by some other path should be at once begun (nutrient enemata, subcutaneous saline infusion, salt-water enemata). Weakness, with cardiac exhaustion, is to be greatly feared, since it will prevent the one therapeutic curative method in the disease, namely, operation.

When the obstruction is in the small intestine, or in the upper part of the large intestine, the patient should be given nutrient enemata. If in the lowermost portion of the large intestine or the rectum, the best one can do is to attempt to prevent threatening collapse by injections of salt or glucose under the skin. In most of the cases the result is unfavourable unless surgical aid is early called in.

4. The Diet in Typhlitis and Appendicitis

By appendicitis we mean those inflammations which start from the appendix vermiformis and either remain localized or extend to the peritoneum or retroperitoneal cellular tissue, or through perforation attack a part or the whole of the peritoneal cavity. A primary typhlitis caused by fæcal impaction or foreign bodies, and often followed by perityphlitis with its symptoms, does occur. With this short statement of my position on an affection which has been the cause of much clinical difference of opinion, I shall close this subject and turn to the dietetic treatment of the condition, reserving the details for discussion in another place.

It is exceedingly difficult to lay down any general rules of diet, because in these affections the food must vary with the character, severity, and course of the disease, as well as with the varying complications and relapses. In stercoral typhlitis the question is easy; in appendicitis it is more difficult. For simplicity, I think it best to adhere to the classification proposed by Sonnenburg, into the catarrhal, the perforative, and the gangrenous forms of appendicitis.

In typhlitis due to fæcal impaction the diet must be so arranged as to aid in the softening and discharge of the fæces. The use of laxatives (castor oil) or of enemata can be advantageously combined with a suitable diet.

In most cases catarrhal appendicitis gets well of its own accord, and the treatment must be so directed as neither to irritate the intestines mechanically nor to increase peristalsis. This is best accomplished by means of a fluid diet, continued until the rise of temperature, the exudate, and the sensitiveness to pressure in the ileo-cæcal region have disappeared. Judging from my experience, I believe that Sahli[8], Quincke[9], and Penzoldt[10] are too extreme when they recommend that for one or more days all food should be at once withdrawn.

After the acute inflammatory symptoms have disappeared, we may for the next week allow the patient, in addition to fluids, to partake of gruels (rice, barley gruels, vegetables in *purée* form, etc.). After another week meat, beginning with the tender varieties, may be added, together with foods made of flour and various stewed and preserved fruits. After four weeks have elapsed the patient may return to his normal diet, which should be so arranged as to counteract the tendency to constipation which is always present in these cases.

In the second variety, the suppurative form of appendicitis, the danger to life is much greater, and the main question is when to interfere surgically. Diet is of secondary importance. Even in these patients I do not believe that an absolute withdrawal of food is advisable, because, on account of the high fever, the patients generally suffer from severe thirst. Penzoldt permits his patients to drink water, and hence I do not see why they may not also take small quantities of tea, milk, wine, and gruels. It seems to me that the fear of a "dangerous stimulation" of peristalsis is exaggerated, all the more as the opium treatment is more thoroughly carried out.* This does not mean, of course, that one should not withdraw all feeding by the mouth where there is severe vomiting or symptoms of intestinal obstruction, and use in its stead frequently repeated nutrient enemata. In general, however, in acute or suppurative stages of the affection, rectal alimentation must be desisted from, if possible, since it necessitates moving the patient about and naturally disturbs the inflamed intestine. If the critical stage of the disease is passed, or if an operation has freed the patient from danger, the diet must be arranged according to the principles above mentioned.

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* [Within recent years, here in the United States, routine opium treatment—formerly so popular—has been almost entirely abandoned, even in those cases treated without operation.—Tr.]

The same general principles apply also to the gangrenous variety of the disease, but the diet must be still more carefully managed. The bowels should be regulated, and by this means the occurrence of relapses prevented. Only in very resistant cases should laxatives be made use of, and one should endeavour to regulate the bowels by means of the appropriate diet for constipation which has already been described.

As the rarer forms of tubercular and actinomycotic appendicitis do not differ essentially from the forms above described, it will be unnecessary to detail the principles of feeding in these cases.

5. The Diet in Intestinal Neuroses

The practical importance of the dietetic treatment of intestinal neuroses is that by its means we may improve the general condition of the patient or remove prominent symptoms of the affection (constipation, diarrhœa, pain, flatulence, etc.). If the diagnosis is absolutely certain, it is questionable whether any special *régime* is necessary at all. Many errors are made in this direction. I have seen, for example, a strict diet cause or increase constipation, tympanites, and colic, which symptoms had presented almost a typical picture of nervous dyspepsia. Such patients tell us that they can not even partake of soup without being troubled with belching or with severe tympanites and abdominal pain. Often the patient will be immediately cured by partaking of a full diet. I would therefore emphatically warn physicians against restricting the diet too quickly when they have patients who complain of intestinal symptoms without discoverable physical signs. In some cases, as in the so-called nervous diarrhœa, this is to some extent necessary. But even in these cases one should in some degree be guided by the sensations of the patient, and should not order all possible sorts of restrictions at once. The patients may make strange assertions which contradict all the acknowledged laws of physiology and pathology, but these statements must not be absolutely ignored.*

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* Thus, I have a patient in whom even the best kind of claret always causes diarrhœa, although Moselle wine promptly counteracts the effect of the red wine.

This class of patients is best treated in a sanitarium, where observation will soon show the appropriate diet for the individual case.

The treatment of nervous flatulence—a condition whose treatment, in the experience of every busy practitioner, gives very unsatisfactory therapeutic results—must be considered in this place. The attempt to determine the cause of the flatulence meets with many difficulties. In more than one case, even after long observation, I have been in doubt as to the nature of the process, and more especially whether or not there was abnormal fermentation in the intestines. Not only do the patients not improve under strict dietetic treatment, but they often become distinctly worse. Tympanites and constipation, when they occur together, may be regulated by an appropriate but not a bland diet; diarrhœa may, however, be accompanied by tympanites, and a constipating diet then becomes necessary. Above all, fluid food should be avoided, as such a diet will make the patient's condition almost unbearable. Very many practitioners are afraid to give so-called gas-forming remedies, but these are often indicated. In the same manner that the sense of pressure in the epigastrium must be relieved by the taking of bicarbonate of soda, so we can often, by means of a diet rich in gas-forming foods, make it easier for the patient to expel gas from his bowel. Many patients of mine who are very sceptical on this point have, to their great surprise, been benefitted by this kind of a diet. But even in these cases individual predisposition must be taken into consideration.

6. Diet in Diseases of the Rectum

Not all affections of the rectum require a special regulation of the diet. Thus, no special diet is necessary in fistulæ, ulcers, or foreign bodies in the rectum. This statement does not mean that in none of the affections of the rectum—especially the surgical diseases—is a regulation of the diet necessary. In some affections of the rectum—stenosis and strictures, hemorrhoids, and fissures—a systematic regulation of the diet is indicated. As regards the first of these affections, the diet to be ordered is the same as that for obstructions of the intestines situated higher up in the alimentary canal.

Special treatment is necessary for hemorrhoids because of the tendency to constipation, alternating perhaps with diarrhœa, and on account of the bleeding which may occur. The bowels should be regulated, and for this purpose the "constipation diet" will usually suffice. Where both patient and physician are impatient, mild laxatives may be used.

Where the tendency to profuse hemorrhages exists, substances which increase the discharge of fluids from the intestines—i. e., acids, alcohol, spices—should, if conditions permit, be excluded from the diet.

The diet in fissures of the rectum and proctitis should be so arranged that the movements are retarded as much as possible; if necessary, opium may be used. Generally this treatment is very satisfactory to the patients because every movement is apt to be followed by severe pain. I keep these cases on a soup diet from eight to ten days. When the fissure has healed and there is no more pain during or after movements, a dose of castor oil should be given. Gradually the patient may be permitted to return to his usual diet. In proctitis the diet should be managed in such a manner that all irritating substances are excluded, and that the movements are soft and easily passed. When the movements are very painful the patient should be placed on a bland diet, and, until the inflammation has disappeared, the intestinal peristalsis should be diminished by means of opiates. Similar treatment is also called for in periproctitis, and is rendered more easy of accomplishment because the appetite of the patient is diminished through the severe pain and the high fever characteristic of the affection. In other cases, a careful emptying of the colon (by means of injections and intestinal lavage) is indicated.

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CHAPTER IX

THE HYDROTHERAPEUTICS OF INTESTINAL DISEASES

In the treatment of affections of the intestines, hydrotherapy plays almost as important a part as in that of the stomach. It is, however, very difficult to give a good account of the action of hydrotherapeutic measures, be the water taken internally or as baths, or both together, or to determine exactly what effects climatic influences have upon the patient. The number of factors that have to be taken into account is so large that in the greater number of cases it is hard to say what part of the results obtained is due to rest and exercise, to diet, to the change in surroundings, or to the treatment of the physicians at the watering place visited by the patient. We are often in the position of the physician who has given a prescription containing four or five drugs; the patient is improved, but the physician is unable to say to which one of the drugs the result was due.

The employment of mineral waters is to a certain extent empirical, for we really have little positive knowledge concerning the action of these waters on the healthy or diseased intestines that we could use as a basis for a systematic treatment. Every busy practitioner, therefore, is accustomed to formulate therapeutic principles for himself according to his experience with these agents. As in my Diseases of the Stomach, I shall also here give an account of the impressions which I have gained from an experience with a large number of carefully observed cases.

For simplicity, hydrotherapeutic measures may be divided into the drinking of waters, hydrotherapeutic baths, seashore, and mountain-air cures. The hydrotherapeutic procedures proper will be considered in the Special Part of this book.

1. *The Drinking of Waters.*—The influence of a course of waters is based upon their action upon the intestine. They may stimulate or diminish peristalsis; they may, so to say, wash out the entire alimentary canal and remove mucus, bacteria, and various toxic products from its interior. The waters may have only an indirect action upon the intestines; for example, in those cases where an improvement of an affection of other organs (gastric, hepatic, or

cardiac disease) is followed by an improved condition of the intestinal functions.

We will limit ourselves to a consideration of the direct action of waters upon intestinal diseases, and shall first give an account of the mineral waters which *increase* peristaltic action. These are:

(*a*) Alkaline carbonated waters (main constituents, bicarbonate of soda and carbonic acid).
(*b*) Alkaline muriated-carbonated waters (main constituents, sodium carbonate and chlorid, carbonic-acid gas).
(*c*) Sodium sulphate waters (main constituent, sodium sulphate).
(*d*) Muriated waters (main constituent, sodium chlorid).
(*e*) Bitter waters (main constituents, sodium and magnesium sulphate).

As regards the first group, they have only a very slight effect in increasing the activity of the intestines. Owing to the sodium bicarbonate and carbonic acid contained in them, they may act as laxatives when given in very large doses. Their use would be limited to very mild cases of constipation, and to those diseases in which a very slight laxative action is desired in addition to the general tonic effect of a mineral water. It need hardly be mentioned that the cold waters have more effect than the warm waters.

Cold alkaline carbonated waters may be arranged, according to their percentage of bicarbonate of soda, into * Vals, Bilin, Fachingen, Fellathalquelle, Preblau, Salzbrunn, Geilnau, and Giesshübl. *Thermal* alkaline carbonated waters are Vichy and Neuenahr.

[In the United States we have the following springs of this class (arranged in the order of their percentage of bicarbonate of soda): *Cold* springs: The California Seltzers, Azula Springs (California), Bladon Springs (Alabama), Adam's Springs (California), and Waukesha (Wisconsin). *Thermal* springs: Ukiah Vichy (California), Howard (Excelsior Spring) (California); and, as an example of the *hot* alkaline carbonated, we have Skagg's Hot Springs in California (temperature = 120° to 140° F.).]

The second group of waters, the alkaline muriated, have a similar action to that of the first group, except that the high percentage of sodium chlorid which some of them contain may make them particularly useful in the treatment of constipation. Of course the temperature of the water has its due influence. Among the *cold* muriated springs I may mention Sczawnicza (Magdalen Spring, 4.61 grams of NaCl per litre), Luhatschowitz (Vincenz Spring, 3 grams NaCl per litre), Gleichenberg (Constantin Spring, 1.85 grams NaCl per litre), Selters (2.3 grams NaCl per litre), Tönnistein (1.4 grams per litre). Among *thermal* chlorid waters may be mentioned Ems (Fürsten Springs, 1 gram NaCl per litre).

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* The detailed account of these mineral waters may be found in my work on the Diagnosis and Treatment of Diseases of the Stomach (Part I, p. 282 et seq., fourth edition).

[The most celebrated springs of this group in the United States are those of Saratoga in New York. In all, there are between twenty and thirty springs at this famous watering place. The amount of sodium chlorid found in them ranges from 1.86 grams (Flat Rock Spring) to 12.04 grams (Champion Spouting Spring) per litre.* Between these two extremes there exists a most varying percentage. Other *cold* alkaline muriated-carbonated waters are those of the Tolenas Springs, California (3.3 grams per litre); the Pacific Congress, California (2 grams); the Dixie Springs, Tennessee (1.9 grams); the St. Clair (Salutaris) Springs, Michigan, the Litton Seltzer, California (each containing about 1.4 grams to the litre); the Ætna Soda Springs, California (0.5 gram to the litre); Manitou, Colorado (0.41 gram); the Medical Lake, Washington State, and the Plymouth Rock, Michigan (each with about 0.26 gram); and the Cresson Magnesia Springs, Pennsylvania, which contains but 0.021 gram to the litre. As examples of *thermal* and *hot* springs of the group under discussion we have the Las Vegas Hot Springs of New Mexico (temperature from 110° to 140° F.), whose largest spring (No. 6) contains 0.26 gram of NaCl to the litre; the Ojo Caliente Hot Springs (temperature 90° to 122° F.), fifteen in number, and containing 0.38 gram to the litre; the Aguas Caliente (Coahuila Valley, California), which contain 0.53 gram to the litre, and whose temperature varies from 58° to 142° F. Finally, there is the Fountain Geyser of the Yellowstone National Park (temperature 179.6° F.), whose water is estimated to contain 0.54 gram of NaCl to the litre.†]

The third group of waters contains a greater or less quantity of *sodium sulphate* and a small percentage of sodium bicarbonate and chlorid, and carbonic acid. *Cold* water springs are Marienbad,

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[* These figures and most of those herein stated are taken from the tables of analyses given in Crook's Mineral Waters of the United States, etc., the latest and undoubtedly the best work on the subject.—Tr.]

[† The waters of these hot springs are employed mostly for bathing purposes.—Tr.]

Tarasp, Elster (Salt Spring), Rohitsch, Franzensbad (Salt Spring). *Thermal* springs are Carlsbad and Bertrich.

The cold sodium sulphate waters have a greater cathartic action than the warm ones. Carlsbad is vaunted for its special good effects on chronic catarrhs of the small and large intestines. Marienbad and the Salt Springs of Franzensbad and Elster are particularly recommended for hemorrhoidal affections and abdominal plethora with constipation.

[Very few of the *cold* springs of the United States of which we have a trustworthy analysis contain a preponderance of sodium sulphate over the other mineral ingredients. Such are the Lineville Mineral Springs of Iowa, which contain 3.1 grams of sodium sulphate to the litre; the Rocky Mountain Springs of Colorado, with 1.8 grams of sodium sulphate to the litre; the Aqua de Vida of California, the Lower Spring with 0.25 gram, and the Upper with 0.3 gram to the litre. The Upper Spring also contains magnesium sulphate. Porter Springs, Colorado, contain 0.47 gram of sodium sulphate to the litre.

The *thermal* and *hot* springs are more numerous. As examples we have the Arrow Head, the San Bernardino, and the Great Paraiso Hot Soda Spring of California; the Pagosa and the Middle Park Hot Springs of Colorado; the Ferris of Montana; and Walley's Hot Springs and Gibson's Mineral Well of Texas. The sodium sulphate in these waters varies from 0.021 gram (Walley's) to 4.4 grams (Gibson's) to the litre.]

The *muriated waters* contain more or less sodium chlorid and a considerable quantity of carbonic acid. *Cold* springs are Nauheim (Kurbrunnen), Neuhaus (Bonifacius Spring), Homburg (Elizabeth Spring), Mergentheim, Kissingen, Soden. *Thermal* springs are Wiesbaden (Kochbrunnen) and Bourbon les Bains.

[Many *cold* waters of this group are found in the United States; some have too high a percentage of sodium chlorid to render them suitable for anything but bathing. Such are the Neptune Spring (Glen Springs, New York), which contains 109.18 grams, and Clark's Red Cross Mineral Well, containing 223.81 grams per litre. The best known of the drinking waters are those of Ballston Spa in New York (United States = 7.29 grams sodium chlorid and Sans Souci = 1.46 grams to the litre); Sheboygan Mineral Well in Michigan, with 5.24 grams; and Glen Springs in New York (Salubria = 3.37 grams; Deer Lick = 1.99 grams). Less generally known, but enjoying an excellent local reputation, are the Louisville Artesian Well (Kentucky), the Lodi Artesian Well (Indiana), the Sweet Springs of Missouri, the Spring Lake Well of Michigan, the Wasatka Mineral Springs of Utah, and the Coronado Springs of California. A number of otherwise excellent muriated waters contain large amounts of sulphuretted hydrogen, so that, strictly speaking, they do not belong in this class. These include the Upper and Lower Blue Lick and the Olympian Springs of Kentucky, the Akesion (Sweet Springs) of Missouri, the Cincinnati Sulpho-saline Spring, etc.

There are fewer *hot* and apparently no *thermal* muriated springs. The Utah Hot Springs (131° to 140° F.), the Yampa (Glenwood Springs) (Colorado), and the Royal George Hot Springs (Colorado), used for bathing purposes, are to be classed with this group of waters.]

The muriated waters improve the appetite, and in large doses increase intestinal peristalsis and thus clear the organism of excrementitious material.

The main action of the bitter waters is cathartic, and hence they may be used to clear the blood and general system of useless substances. We may mention Franz Joseph Spring in Buda-Pesth (24 grams of magnesium sulphate and 23 of sodium sulphate per litre), Hunyadi János in Buda Pesth (16 grams of magnesium sulphate and 15 of sodium sulphate per litre), Pülna in Bohemia (12 grams of magnesium sulphate and 16 of sodium sulphate per litre), Friedrichshall (5 grams of magnesium sulphate and 6 of sodium sulphate per litre), Apenta, etc.

[There are between twenty and thirty cold and thermal bitter-water springs in the United States. Although none have as high a percentage of magnesium or sodium sulphate as the above-mentioned European waters, still many of them are excellent cathartic waters. At the head of the *cold* springs stand the B. B. Mineral Springs of Missouri, which contain 11.5 grams of magnesium sulphate and 1.04 of sodium sulphate per litre; the Estill (Purgative) Springs, 4.5 grams of magnesium and 1 of sodium sulphate; the Epsom Spring (Crab Orchard, Kentucky), 3.5 grams of magnesium and 1 of sodium sulphate per litre; the Harrodsburg Springs, Kentucky (Saloon Spring = 3.8 grams and Grenville = 2.2 grams magnesium sulphate per litre); Sowder's Spring (Crab Orchard) has 3 grams of magnesium and 0.4 of sodium sulphate; Foley's Spring, 3.4 grams of magnesium and 1 of sodium sulphate; Mono Lake (California) contains 2.1 grams of magnesium and 0.3 of sodium sulphate; the American Carlsbad Springs [*] (Illinois), 1.8 grams of magnesium and 0.1 of sodium sulphate, and the Gypsum Springs of Arizona have 1.7 grams of magnesium with 0.5 of sodium sulphate. The remaining springs contain magnesium sulphate in amounts varying from 0.02 gram to 0.9 gram to the litre. Mentioned according to their percentage of magnesium sulphate, they are the Alleghany Springs of Virginia, the Mineral Park Bitter Spring (Arizona), the Greenbrier (West Virginia), the Bedford (Magnesia and Bowling Alley) Springs (Pennsylvania), Seigler's (Magnesia) Spring (California), and the Mountvale Springs (Tennessee). Others charged, however, with sulphuretted hydrogen are the Avon Sulphur and Sharon Springs [†] of New York, the Salt Sulphur Springs of West Virginia, the French Lick Springs (Proserpine and Pluto) of Indiana, and the Indian Springs of Georgia.

As examples of *thermal* and *hot* bitter waters we cite the Soda (drinking) Spring (Virginia Hot Springs), temperature = 74° F.; the Idaho Hot Spring (Colorado), temperature = 85° to 115° F.; the numerous Catoosa Springs of Georgia, temperature = 212° F.]

There are some mineral waters which diminish peristalsis. All the waters mentioned above, except the bitter waters, when they are taken warm and in small doses, have this action. Especial mention must be made of Carlsbad Sprudel water, which has a great reputation in chronic diarrhœa. In addition, the waters which contain lime and iron must be considered in this connection.

Of the lime waters, particularly those which contain calcium carbonate, Germany has the following varieties: Wildungen (König's Spring), Driburg (Herster Spring), Lippspringe (Arminius Spring), Rappoldsweiler (Carola Spring), Marienbad (Rudolf's Spring), Coburg (Mariannen Spring); the last-named water is generally used for exportation. The table below gives the composition of these waters:

	Carbonate of lime and carbonate of magnesium in litres.	Free CO_2. Cubic Centimetres.
Wildungen (König's Spring, temperature 10° C.)...	2.23	1,322
Driburg (Herster Spring, temperature 10.6° C.)...	1.51	1,043
Rappoldsweiler (Carola Spring, temperature 18° C.)	0.77	
Lippspringe (Arminius Spring, temperature 21.2 C.)	0.61	646
Coburg (Mariannen Spring)......................	0.3463	343

[* The name "Carlsbad" is inappropriate, for the waters of Carlsbad in Bohemia contain large quantities of sodium sulphate, and no magnesium sulphate whatever.]

[† This is essentially a sulphuretted and sulphate of calcium spring.]

[The list of carbonate of calcium springs in the United States is too long to be even detailed here. A number of them contain considerable quantities of this salt. All the Saratoga waters have it in amounts ranging from 0.72 gram to the litre (Empire and Eureka Springs) to 2.9 grams (Geyser Spouting and Hathorn) and 3.9 (Champion Spouting).* Other waters of this class are the Americanus Mineral Well (formerly Michigan Congress), with 1.47 grams per litre; California Seltzer, with 1.25 gram; Montesano (Missouri), 1.2 grams; (Tolenas (California), 0.85 gram; Sans Souci Springs (Ballston Spa, N. Y.), 0.74 gram; Akesion (Sweet Springs, Mo.), 0.69 gram; and the Soda (Summit Springs, Cal.), 0.65 gram to the litre. To mention a few others with lesser amounts of calcium carbonate, there are the Geneva Lithia, the Adirondack, and the Avon Sulphur (Lower) Springs of New York; the Kickapoo and Indian Springs of Indiana; the Sheboygan and Leslie of Michigan; the Boiler (bathing) and Soda (drinking) of Virginia Hot Springs; the Royal George of Colorado, Waconda of Kansas, Crocker of Tennessee, Old Sweet of West Virginia, the Upper and Lower Blue Lick of Kentucky, the Bartlett of California, etc.]

The Iron or Chalybeate Waters.—These contain carbonates as well as sulphate of iron, and have a tendency to diminish secretion in the body. They are especially useful in chronic diarrhœa or intestinal catarrhs with diarrhœa, of which anæmia is the causative factor. The best known carbonated chalybeate waters are Franzensbad, Elster, Driburg, Reinerz, Rippoldsau, Pyrmont, Schwalbach, and Cudowa. Of sulphate-of-iron waters we may mention Ronneby in Sweden, Levico and Roncegno waters, and the Guber Spring.

[Many waters of this country have been found upon analysis to be very good chalybeates. Combining, as many of them do, a goodly proportion of other minerals (sodium, magnesium and calcium, chlorids, carbonates, and sulphates), they are seen to be most excellent mineral waters. But a limited number can be mentioned; an analysis and other details will be found in works devoted to the subject of mineral waters (Crook, A. N. Bell[2], Walton[3], etc.). Named in the order of their percentage of carbonate of iron, we have the Iowa White Sulphur Springs; the Chittenango Sulphur Springs of New York (Magnesia Spring); the Owasso Spring

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[* In addition to the amount of calcium carbonate here stated, the Saratoga, as well as all the other waters of this class, contain considerable proportions of magnesium carbonate.—Tr.]

(Michigan); the Pacific Congress (California); the Sparta Mineral Wells, Wisconsin (Magnetic Mineral Well); Napa Soda Springs and Mono Lake of California; Fruit Port Well (Michigan); Saratoga Springs, New York (Putnam, Columbia, Hamilton, Excelsior, and Eureka Springs); Wilhoit's Soda Springs (Oregon); Ballston Spa (Sans Souci) and the Adirondack Spring of New York; Arrington Springs (Nos. 1 and 2), Kansas; Americanus Mineral Well (Michigan); Massanetta Springs (Virginia), etc.

The following are the principal waters containing the sulphate of iron; they are arranged according to amount of that salt found in them: The Overall Mineral Wells (Nos. 2 and 1) of Texas; the Oak Orchard Acid Springs (No. 2) of New York; Gaylord and Garlick's Mineral Springs of Pennsylvania; Bath Alum Springs (No. 2) of Virginia; Indian Springs (Indiana); Bedford Alum Springs (Virginia); Texas Sour (or Caldwell's) Springs; Austin's Springs (Tennessee); Iron Lithia Springs (Virginia); Wilbur's Springs (California); Fauquier's White Sulphur Springs (Virginia), etc. In addition, sulphate of iron has been found in a number of the California geysers in amounts ranging as high as 0.49 gram to the litre.

Free sulphuric acid is found in the Oak Orchard Springs (about 2.25 grams per litre); the Texas Sour Springs (0.124 gram per litre); the Gaylord and Garlick, the Iron Lithia, and Bath Alum Springs (about 0.086 gram per litre); the Bedford Alum (about 0.068 gram per litre); and owing to its acid reaction is probably also present in the Overall Mineral Wells (Crook).]

In some cases—I believe in far too few—enemata of mineral water are made use of. For this purpose the thermal waters of Carlsbad, Ems, Wiesbaden, and Neuenahr are especially appropriate, particularly if there exist a catarrhal affection of the lower segments of the intestines. Pollatschek[1], more than any one else, has reported very favourable results from small enemata (100 to 200 cubic centimetres) of warm mineral waters in chronic catarrh of the large intestine and in ulcerative processes and intestinal neuroses. I should think that small quantities of chalybeate waters per rectum might be useful in these cases in which iron is not well borne by the stomach.

If, after this brief *résumé*, we next ask ourselves what practical benefits are to be derived from the use of the above-named mineral waters, we shall find it very difficult to answer our question satisfactorily.

As already mentioned, so many subsidiary factors are to be taken into account that it is difficult, if not impossible, to clearly define their individual action. Notwithstanding this, the following statements are probably correct. To simplify matters let us classify the most frequent intestinal affections as chronic catarrhs of the large and small intestines, chronic constipation, and chronic diarrhœa. It would be correct also to distinguish between mild and severe types, but such a division depends largely upon subjective impressions.

In the milder catarrhs of the small intestine I have seen excellent permanent as well as temporary results from the thermal waters of Carlsbad. These cases are unsuitable for the cold sodium sulphate, the muriated, or the carbonated springs. The warm Carlsbad waters are also most beneficial in mild catarrhs of *the large intestine*, associated with constipation or with constipation alternating with diarrhœa. Next to these waters, the cold sodium sulphate and the warm and cold muriated waters give the best results. Good results are sometimes obtained from the waters of Ems.

Positive benefit may be expected in hemorrhoids from the use of the Glauber salt waters (particularly Marienbad), as well as from the cold muriated waters (especially Kissingen, Homburg, and Mergentheim). The effect is frequently permanent; usually, however, the course of waters must be repeated. Water treatment in mild cases of habitual constipation is fairly satisfactory. These are sometimes permanently relieved by the sodium sulphate and muriated waters, but generally the relief is only temporary.

Improvement may be expected in chronic diarrhœa from the use of the Carlsbad waters (the Sprudel in small amounts), the muriated waters (small doses warmed), and from the waters of Ems.

The above-mentioned calcareous waters are very useful in chronic diarrhœa, although, strange to say, they are very little used for this purpose. In combination with a suitable diet I obtain good results from their use. From an extended personal experience I can warmly recommend the Marienbad Rudolfsquelle, the Coburg Mariannenquelle, and the Lippspringer Arminiusquelle.

Severe forms of intestinal catarrh, constipation, and diarrhœa are less likely to be benefitted by a course of mineral waters. *The results are either temporary or entirely negative.* Of the many hundreds of private patients who have drunk the sulphated or the muriated waters at the original springs, I have not yet seen one who has derived any permanent benefit from the waters themselves.

I could note an improvement only in those patients who had other treatment, and for whom, therefore, the drinking of the waters was really superfluous.

The cause of failure in these cases is very clear. Proper regulation of the diet is the main factor in the treatment of the severer types, and not even in a place so well organized as Carlsbad is this feature as thoroughly attended to as is necessary. Rest in bed for a longer or shorter period is essential to the cure of the severer forms of chronic diarrhœa and of the intestinal catarrhs that cause them. It is very difficult or even impossible to obtain this in a watering place, unless the latter contains a sanitarium such as have of late been successfully introduced in various watering places. From this it follows that only mild and very recent cases are adapted to a course of water treatment; all others should be treated in a sanitarium or a hospital. Since a number of mineral watering places (e. g., Kissingen, Wiesbaden, Carlsbad, Baden-Baden, etc.) have now well-conducted private institutions in which proper regulation of the diet can be, wherever necessary, combined with mechanical therapeutic measures such as massage, intestinal irrigations, etc., we can now send even severe and protracted cases away. There are more such private sanitaria throughout Germany and Switzerland, but as it is impossible to commend them all in this place we refrain from mentioning any.*

Is the benefit to be derived in mild cases sufficient to warrant a patient in moderate circumstances incurring the heavy expenses of such a cure? Where a permanent cure can not be anticipated—and this is true in the majority of cases—it is best to advise against such treatment. With the wealthier classes it is quite different, for they visit the baths every year, either from custom or in order to recuperate from the trials of winter society. It matters, therefore, very little to them whether they derive any benefit from the course of waters or not. At all events, it is wrong to promise any patient a permanent cure of his intestinal trouble from a four weeks' course at a mineral spring. Unfortunately, the physician is too optimistic regarding the effects of mineral waters, and it is not surprising, therefore, that failure to obtain such results is explained upon the ground that "the wrong spring was selected."

Mineral waters are often drunk at the home of the patient. This is advisable, if at the same time it can be combined with proper dieting and also a certain amount of rest and freedom from daily occupation. It is also useful as a preliminary test in doubtful cases.

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* A fairly complete list will be found in Penzoldt-Stintzing's Handbuch d. speciellen Therapie innerer Krankheiten, 1. Aufl., Bd. iv, S. 274.

2. *Baths.*—Very often baths are given in connection with a course of mineral waters and may even constitute the main part of the treatment. They are classed as natural baths, iron, salt-water, and mud [" moor "] baths. The last-mentioned variety are very important in the treatment of chronic exudative processes about the intestines. The mud baths of Marienbad, Franzensbad, and Elster are the most popular ones.* Salt-water baths are found inland in low as well as in mountainous regions (Reichenhall, Kosen, Salzungen, etc.) and at the seashore (Colberg, Swinemünde, etc.). There can be no doubt as to the favourable influence of these baths upon many cases. Thus in perityphlitic exudations I have occasionally observed remarkable benefit follow the use of salt-water baths. For a combination course of mineral waters with salt-water bathing Kissingen and Wiesbaden are good; for a course of waters with mud baths, Marienbad, Elster, Franzensbad, and Pyrmont are best.

3. *Climatological Treatment* (Seashore and Mountain Air).—This may be recommended alone, or may also be combined with a course of mineral waters. Without doubt climatic treatment is best adapted to nervous affections of the intestines. It is very often also, and quite correctly, advised as an after treatment to a course of waters. The physician will very frequently have to decide for his patient between seashore and mountain air. This question can only be considered for the individual case; very frequently, indeed, equally valuable results may be obtained from either region. As a rule, patients whose ordinary vocation inclines them to sedentary habits should be sent to the mountains; while those living in large inland cities and accustomed to excitement and running about, and to gross irregularities in eating and drinking, should be sent to the seashore. On the other hand, those suffering from organic intestinal disorders should not be sent to the seashore or to the mountains. I would warn against permitting patients suffering from chronic intestinal catarrhs to partake of *table d'hôte* meals such as are ordinarily served at the seashore or in the mountains. As already mentioned, it is often possible to combine cold-water treat-

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[* In the United States mud baths are given, among other places, at the Arkansas, Virginia, and Las Vegas Hot Springs. At these resorts elaborate bathing establishments, modelled after those in vogue at the most celebrated European watering places, have been established.—Tr.]

ment with a sojourn in the mountains, and in suitable cases we ought to take advantage of this. Owing to the enormous number of seaside and mountain resorts in Germany and in foreign countries, it is impossible to give even a partial list of them.

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CHAPTER X

MASSAGE, ELECTRO- AND HYDROTHERAPEUTICS IN INTESTINAL DISEASES

1. Massage

In intestinal diseases massage is employed to stimulate the relaxed muscles of these organs, and to cause diffusion and absorption of local inflammatory and peritoneal processes.

The most frequent indication for massage is furnished by atony of the intestines.

TECHNIC OF INTESTINAL MASSAGE *

In order to relax the abdominal walls we begin with *rotary effleurage* (stroking). In stout persons this is followed by *petrissage* (kneading), the object being the crushing of individual fat globules. Petrissage of the deeper tissues is the most important manipulation in intestinal massage.

"With both hands we grasp the intestines through the abdominal walls, and as through we were handling a muscle that we wished to express, we make the same zigzag forward and backward movements that we would in kneading such muscle. We thus proceed over the entire abdomen. The next step is to stroke the fæcal masses out of the intestines. We begin with the ascending colon, pass upward, follow to the left along the transverse colon, and then continue downward until we reach the sigmoid flexure. At first, stroking movements are made; later, rotary petrissage is substituted, for this cts more energetically in mechanically removing fæcal contents." Hoffa describes "rotary petrissage" (kneading) as follows: "With the hands held at an acute angle to the abdominal wall, and with the finger tips of the left hand resting upon those of the right and directed upward toward the chest, rotary movements are made in the ileo-cæcal region [Fig. 22]. Since sudden pressure would result in rigidity of the abdominal walls, the rotary movements must be gentle, the deeper parts being very gradually approached. The movement is the same that we make in palpating deep-seated abdominal tumours. The patient is told to breathe deeply, and with each expiration we press deeper. The advancing movement on the part of the operator proceeds directly from the shoulder joint, while the finger, wrist, and elbow joints should be held almost rigid."

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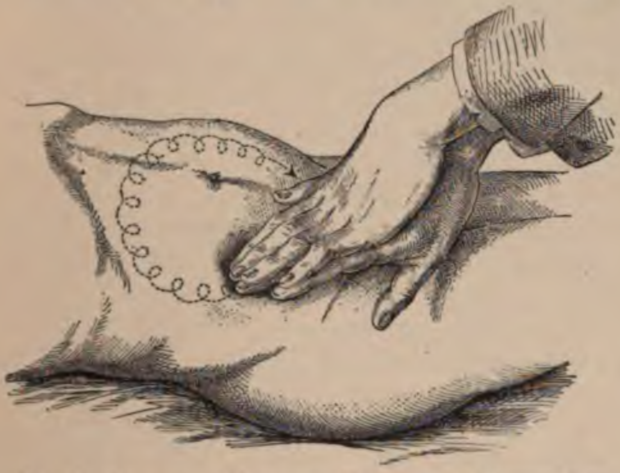
* We here follow the description given by Hoffa in his Technik der Massage (Stuttgart, 1893), to which work the reader is referred for all further details. (Com- also the section on Massage in my Diagnostik und Therapie der Magenkrank- Part I, p. 324, fourth edition.)



[FIG. 22.—POSITION OF HANDS AND DIRECTION OF MOVEMENTS IN INTESTINAL MASSAGE (Hoffa).—TR.]

Tapotement (*percussion*) *and vibrations* of the abdominal wall follow the deep petrissage. These tend to stimulate the smooth muscles of the intestines, and to promote alterations in the circulation in the blood-vessels.

Besides atony of the intestines (especially that associated with flatulence), massage is beneficial in localized peritonitic processes about the intestines, particularly in perityphlitis or appendicitis after the acute stage has passed. In these cases the greatest care must be taken. Above all, we should make it a strict rule never to begin massage until weeks after all inflammatory manifestations have disappeared. The regions of the cæcum must no longer be sensitive to pressure. It is hardly necessary to state that if there is the least suspicion of an abscess or of tubercular appendicitis massage should never be attempted.

This last precaution applies also to new growths of the intestinal tract, benign as well as malignant. I would also warn against employing massage in intestinal obstruction. As a rule, it can only add to the gravity of the condition.

Each sitting should last ten minutes, and no single manipulation should require more than two or three minutes. The treatment should first be conducted daily for four weeks; after that every two or three days for four to six weeks. In constipation the results from massage will vary largely, for they depend upon the degree of the intestinal difficulty, as well as upon the patience of the subject, and above all upon the skill of the *masseur*. My own experiences in the matter have led me to form the following conclusions: A permanent result (i. e., regular and sufficient intestinal evacuations) can be obtained from properly conducted massage combined with therapeutic measures which tend to bring about the same result—i. e., diet, gymnastics, electricity, rowing, turning, and other bodily exercises.

On the other hand, in some cases of intestinal atony I doubt very much whether massage alone can give more than temporary relief. Very soon after the discontinuance of the massage, even though applied for weeks by an experienced person, the intestine usually fails to properly perform its functions. This result is quite contrary to that obtained from the above-mentioned diet (page 000), where in the vast majority of cases a permanent effect is secured, continuing even when the diet *régime* is no longer strictly followed. We do not deny that exceptions do occur, but these do not alter the general rule.

Regarding massage in localized peritonitis, I can say but very little, for my experience has not been extensive. I am, however, in favour of a trial under the above-mentioned precautions. At times considerable benefit may be derived from the combination of massage with indoor gymnastics, such as have been introduced by G. Zander. This is not the place to enter into a description of such gymnastics, especially as they can only be learned through practical experience.

2. Electrical Treatment

This includes the use of the faradic, the galvanic, and the galvano-faradic (mixed) currents. The application, as in the stomach, may be either external or intra-abdominal, or, more correctly speaking, rectal. At present opinion is very much divided as to whether galvanism or faradism, or their combination, is more beneficial, and also as to whether the extra-abdominal or intrarectal method gives the best results. In fact, some authors regard the application of electricity to the intestines as utterly useless.

Our scientific knowledge regarding the action of the electric current upon the intestines is very limited. We know that visible and palpable intestinal movements can be produced in persons with thin abdominal walls or with inguinal hernia (von Ziemssen). In a case of peristaltic restlessness under my observation we could detect a temporary increase of the intestinal peristalsis. The only systematic experiments were those of Schillbach[1] upon exposed rabbits' intestines kept in a bath of normal salt solution. His results would seem to indicate that the faradic and the galvanic currents differ in their effects upon intestinal peristalsis, inasmuch as a faradic current of medium strength causes—particularly in the small intestine and less so in the large—ascending and descending waves of contraction of a few centimetres of the stimulated bowel. With the galvanic current he observed localized contractions at the cathode, and peristaltic waves of contraction at the anode. In view of these observations, Leubuscher[2] and Schillbach particularly recommend the employment of intrarectal galvanization (anode on the abdominal wall, cathode in the rectum), and from their experience they believe galvanization gives better results than faradization. There is considerable discrepancy between the views of authorities on the efficiency of electro-therapeutic measures. Nothnagel[3] considers electrization of the intestine less beneficial than massage; Ewald[4], too, sees little or no use from electricity in intestinal diseases. On the other hand, Eichhorst[5] reports a case in which unsuccessful attempts were made for eight days to secure a movement of the bowels, and in which a single application of the faradic current was followed by copious evacuations and complete cure of the patient. Unfortunately, the nature of the obstruction (fæcal impaction ?) can not be determined with certainty from the published reports of these cases.

I should like to describe the technic of intestinal electrization before speaking of the results gained from my own experience. For external application either broad electrodes are to be employed or electric rollers or brushes. As in the electrical treatment of the stomach, so in intrarectal electrization, I have many years employed a soft-rubber tube, with numerous perforations near its tip and with a platinum spiral in its interior (Fig. 23). In addition to the switch for making and breaking the current, the upper part also is provided with a small tube for the in- and outflow of water. The filling of the rectum is accomplished in the usual manner by a funnel and rubber-tube attachment. From 100 to 200 cubic centimetres of lukewarm water are allowed to flow into the bowel. The other electrode is placed upon the abdomen and is moved along the entire course of the large intestine. Each application should last ten minutes. The faradic current can be applied strongly enough to produce distinct visible muscular contractions; the patient should experience a definite and barely painful prickling sensation. Twenty to thirty milliampères of the galvanic current will suffice.

My personal experience is limited almost entirely to intrarectal faradization. My conclusions are to be considered all the more trustworthy because I resort to intestinal electrization only when other means have failed (i. e., in the most obstinate cases), and I seldom combine different methods of treatment. *I can state that intrarectal treatment succeeds in many of the cases in which nothing at all could be accomplished by massage, diet, or gymnastics.* The patients have well-formed movements daily. We can exclude any possible action from the water, for, aside from the fact that it is withdrawn through the tube at the termination of each electrical application, those cases of constipation in which I employ intrarectal faradization are of such an obstinate nature that small enemata of water have no effect whatever. Besides this, defecation does not immediately follow rectal faradization, but frequently occurs only after many hours. In some cases a permanent benefit is obtained, in others the patients are compelled for months to use the electric rectal sound. Aside from the so-called auto-massage, which is hardly deserving of serious attention, electrization has the advantage over massage that any intelligent patient can employ it for months on himself.

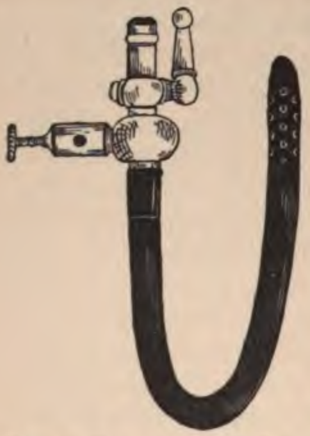


FIG. 23.—ELECTRIC RECTAL TUBE.

The use of the electric current is therefore generally indicated in paresis of the intestinal muscle; its most important sphere, however, lies in those forms of habitual constipation in which over-distention of the ampulla recti had gradually led to paresis of the rectal muscles and sphincters. In this condition, skilfully applied electrical treatment will, as I have repeatedly convinced myself, result in permanent benefit to the patient.

3. Hydrotherapeutic Measures

In diseases of the intestines, use is made of external and internal hydrotherapeutic measures. The internal ones are mostly limited to the use of water in the rectum, and owing to their great practical importance will be treated of separately elsewhere. In the following we will consider external hydrotherapeutics only. Of these we employ:

1. *Moist Applications.*—These may be cold, warm, so-called "Priessnitz" (moist) applications and poultices. The former class exert a stimulating, the latter a sedative, action on the bowel. The Priessnitz pack while cold stimulates, and later exerts a sedative influence on the gut. Hot bran poultices relieve pain, probably owing to the fact that the heat causes a momentary paralysis of the sensitive nerve-endings. Hot or cold water bottles or water bags may be used instead of moist applications; in general practice, clay bottles filled with hot water and wrapped in flannel or some such impermeable material suffice. In place of hot bran poultices, I have long used pieces of felt dipped in hot water and then covered with an impermeable material.

2. *Baths.*—These are classed as full baths, half baths, and sitz baths, all of which can be given at varying temperatures and combined with other hydriatic measures.

3. *Douches.*—These are given in the form of cold or warm or of alternating hot and cold (Scotch) douches. They are classed as *shower*, *needle*, *jet*, and *vapour douches.*

4. *Cold or Lukewarm Applications, with Friction, etc.*—In most cases these different procedures are combined in various ways, and frequently electricity or massage is given in connection with them. It would be out of place to enter into a detailed description of these here. External hydrotherapeutic measures are mainly indicated in chronic and benign intestinal affections—i. e., catarrhs and neuroses. Intestinal neurasthenia in particular is often very much benefitted by hydriatic treatment. At the present day hydrotherapeutic treatment is applied almost exclusively in institutions, and its results are in part due to the water applications, but more so, in many cases, to the personality of the conductor of the institute. It can be readily

understood that those institutions are best which provide an individualized diet *régime.*

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CHAPTER XI

INJECTIONS (ENEMATA, INTESTINAL LAVAGE, AND DOUCHES), INFLATION, AND GASTRIC LAVAGE IN INTESTINAL DISEASES

1. Injections (Enemata, Intestinal Lavage, and Douches)

INJECTIONS are employed for various purposes:

1. To stimulate peristaltic action, or by softening hardened fæces to facilitate their removal.

2. As intestinal lavage, (*a*) for the removal of pathological substances (mucus, blood, and pus); (*b*) for the purpose of irrigating the mucous membrane with watery solutions of medical substances.

3. Mechanically to remove concrements and foreign bodies.

4. As douches, to strengthen and stimulate the paretic intestinal muscle.

Technic.—Nowadays enemata are given either by means of an irrigator consisting of a reservoir of varying material (rubber, glass, or metal) connected by rubber tubing, the so-called rectal tube; or else by means of Hegar's apparatus, in which the reservoir is formed by a glass or rubber funnel. The irrigator is better adapted for use by the patient himself than is Hegar's apparatus, but the latter is far more preferable for the giving of an enema by a second person, since it permits him to regulate the pressure, and in case of too much distention, by simply lowering the funnel, to let any desired amount of water or air out of the intestine. For auto-irrigations I recommend an irrigator holding 3 litres, which is fastened to a support, and, like the well-known Leube-Rosenthal apparatus for gastric lavage, is also furnished with a T-shaped tube. According to Quincke, the rectal tube is best made of soft rubber; the English [French] stiff or hard-rubber tubes are decidedly inferior to it, and if employed at all, should be thick and rounded off at their lower end and provided with two or three large [lateral] openings. To avoid injury to the parts, all rectal instruments should be smooth and free from cracks. For this reason soft-rubber tubes are to be preferred.

In place of the above-mentioned apparatuses there are others adapted to auto-injections per rectum (e. g., the so-called injection pumps, alpha syringes, etc.), all based upon the principle of forcing water into the rectum under a certain degree of pressure. All in all, the value of these and similar apparatuses described in the catalogue of instrument makers is not very great. Even their advantages for use during travel is questionable. I would emphasize, better results are not obtained with these than with the irrigator or funnel. As regards the technic of giving or taking an enema, in the first place, one must have a definite idea of the object in view. If it be desired to act upon the colon in order to stimulate peristalsis, this is best done with the rectum as empty as possible. The method employed is similar to that used in giving an intestinal douche, to the discussion of which we refer the reader for technic and indications.

For the removal of hardened fæcal masses from the intestine, we employ thermal, mechanical, chemical, or electrical methods, singly or combined.

Thermal stimuli consist in the injection of cold fluids of a temperature of 18° C. [65° F.] or less. Sometimes irrigations of ice water are recommended—e. g., in bleeding from hemorrhoids—but such measures are not without danger.

The mechanical effect is obtained, in the first place, from the introduction of the instrument itself, and, secondly, from the injected water.

The purpose of chemical agents is either to exert an influence upon the intestinal wall or to soften hardened fæces.

The effect of intrarectal electrical treatment has already been described on page 174.

It is not easy to state which of these methods is the best. In some cases—i. e., in certain stages of constipation—one apparently obtains good results from any one of them. Without doubt, individual idiosyncrasies play an important rôle here just as they do in the medicinal treatment of constipation. I shall not, therefore, enter into a discussion of the relative value of the various methods in use, but shall confine myself to those which my experience has taught me are best.

The quantity of fluid that should be injected into the rectum is a question of primary importance. It is clear that 1 or 2 litres of fluid, when injected into the lower portion of the bowel, will cause an acute extreme distention of the intestine and a resulting tenesmus, so that the greater part or the whole of the fluid is expelled without sufficient purgative action. It is immaterial whether one uses cold or warm water, oil, vinegar, soapsuds, or glycerin. Even the more active irritant remedies exert an insufficient influence upon the bowel when they remain in it for a short time only. If the lowermost portion of the intestines contain fæcal masses, these will be forced out mechanically with the water.

The general rule to be followed in the majority of cases is this: the quantity of fluid used should be small, not exceeding 300 c. cm., and should be slowly injected under slight pressure. It is best to have the patient lying on the left side with the pelvis raised. This amount of fluid should be retained for several hours, so as to cause a gradual softening of the intestinal contents or a stimulation of the smooth muscle fibers of the bowel wall. I generally leave orders to have these enemata given at night, so that the bowels move the next morning, or else given in the morning, so as to obtain a movement in the evening. Glycerin, soap, sugar, honey, etc., added to the enema increase the softening effect upon the fæces. Penzoldt's[1] experience, which I can substantiate throughout, was that soap acted most energetically in this respect, and oil somewhat less so. Others have recommended limewater for the same purpose. As a rule, I order the following as an effectual enema: One teaspoonful of soap shavings, or of good glycerin soap, dissolved in a quarter of a litre of lukewarm water to which is added one to two tablespoonfuls of glycerin. As a result of Fleiner's[2] recommendation, oil injections of from 400 to 500 c. cm. are very frequently used. Fleiner believes that it is very important to use only the purest oil, and for this purpose he has especially recommended sesame oil. The disadvantage of the method is that frequently the patient's clothes are soiled by the oil. For this reason, and also because the oil partly adheres to the walls of the irrigator and tube, I order the oil to be used in the form of an emulsion, and recommend the following procedure: A piece of soda the size of a bean is dissolved in a quarter litre of water; to these are gradually added two tablespoonfuls of commercial cod-liver oil and the mixture thoroughly shaken; two tablespoonfuls of castor oil are then added, and the whole mixture shaken until an emulsion has been formed. The resulting emulsion readily flows through the irrigator, is well retained by the patient, and even in the most obstinate cases will succeed in softening hardened fæces. In an experience of almost ten years I have never seen any irritation, pain, or other unpleasant symptoms from them, and I therefore warmly recommend them.

In this place it is proper to discuss "high enemata," concerning which much is said in daily practice, and to the mechanism of which due consideration is seldom paid. We have already noted that rectal tubes can seldom be introduced beyond the sigmoid flexure or the descending colon, since they are generally arrested in the sigmoid flexure, and further attempts result in a bulging of the intestinal wall. It is no better with rigid French or English tubes than with the soft Nélaton tubes; the former pass more readily into the sigmoid flexure, although they are very apt to be arrested by the folds of this portion of the intestines. We see, therefore, that the whole question of the so-called high enemata is in many respects an illusion.

Even for extensive lavage of the upper portions of the intestines it is not necessary to introduce the tube very high. By means of auscultation one may convince himself that even when the tube lies in the rectum and the patient is properly placed (lying on his [left] side with raised pelvis, or in the knee-chest position), fluids pass as far as the cæcum.

Irrigations of the bowel by means of a funnel are especially useful for the removal of fæcal masses and tightly adherent mucus from the large intestine. In obstinate constipation I have had very good results from enemata consisting of several litres of soapsuds (with the addition of glycerin, castor oil, and cod-liver oil if necessary).

Indications for Enemata and Irrigations of the Intestine.—Enemata are most frequently used in acute and habitual constipation. Regarding their usefulness or disadvantages there is a diversity of opinion among physicians and laymen. We can not here enter into this question. From my own experience, I believe that enemata have a wide field of application both in acute and in habitual constipation; when properly given they are generally successful. They are said to have the disadvantage of distending the large intestine and thus increasing its atony. This is certainly true where very large enemata are given. When given as recommended by us this result need not be feared. Therefore, whenever possible in habitual constipation enemata should be given preference to laxatives. An internal laxative is best in acute constipation complicated by acute gastric catarrh. The second most frequent indication for enemata is chronic catarrh of the large intestine with profuse diarrhœas or excessive mucus formation. Here one should use lukewarm water, or, better still, physiological salt solutions or solutions of medicinal agents. The latter should be chosen according to the effect desired; thus we may use antiseptics such as lysol, boric acid, salicylic acid, salicylate or benzoate of soda, thymol, etc., or astringents such as tannin, aceto-tartrate of aluminium, and nitrate of silver,* or mucus solvents such as boracic acid, or bicarbonate, carbonate, or acetate of soda,† or limewater. Here intestinal lavage is much better than enemata, for it enables us to first cleanse the bowel and then immediately thereafter apply our medication. If tenesmus occurs, it may at once be allayed by lowering the funnel. The lavage should be repeated once or twice daily.

A further indication for lavage is furnished by chronic stenoses, where these are not caused by extensive ulcerations or tumours which tend to perforate. Since we have no general diagnostic criterion for these conditions, great care must always be exercised—i. e., avoid great pressure, and give only small enemata, frequently repeated. Syringes which force fluids into the gut should never be used. While the effect from these irrigations is mainly palliative, skilful application may occasionally enable us to relieve a sudden and dangerous total intestinal obstruction arising from dietary error—e. g., impaction of indigestible food remnants.

It is sometimes possible to loosen and wash out gallstones, coproliths, or swallowed foreign bodies causing an obstruction. In obstruction from gallstones I would recommend the employment of injections of chloroform water (10 : 1,000) in order to dissolve small fragments of these concrements. This should be done under anæsthesia, as it is then possible to pass beyond the ileo-cæcal valve. I have not had any practical experience in the matter.

Furthermore, enemata are indicated in acute and chronic invaginations: in the acute, in order to reduce the invagination; in the chronic, to prevent or relieve coprostasis above the invaginated portion. Nothnagel especially recommends injections of 5 to 8 per cent saline solutions, and bases his recommendation upon animal experiments, in which he found that physiological invaginations are overcome by saline injections.

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* We use one teaspoonful of tannin, boric acid, and aceto-tartrate of aluminium, or one half to one gram of silver nitrate to the litre of water.

† Bicarbonate, carbonate, or acetate of soda are used in the strength of one dessertspoonful to the litre.

Intestinal Douche.—In the intestinal douche we have a means of stimulating and strengthening the neuro-muscular apparatus of the lower portion of the large intestine. Here, as in the stomach, we employ a funnel and rubber tube armed with a short, soft, Nélaton rectal tube, the end of which contains ten to fifteen small openings. The tube is introduced as far as possible, preferably under the guidance of the finger. Ordinary cold water, or cold water alternating with hot, is employed, or we may also use water charged with CO_2. As already mentioned, it is best to have the rectum emptied before giving the douche.

In a series of cases of atony of the large intestine, especially in those in which scybala were retained in the recesses of the sigmoid flexure and the ampulla of the rectum and caused marked tenesmus, this procedure has proved a very valuable one.

2. Inflation of Air

Inflation of air, the diagnostic value of which has already been discussed, is warmly recommended by some writers as a means of relieving obstructions, especially of the lowermost portions of the intestine. Curschmann[3], in particular, has often spoken in favour of intestinal insufflation in cases of volvulus of the sigmoid flexure and intestinal stenoses, and he prefers it to the injection of water. He recognises, however, that there are dangers connected with the procedure, and therefore advises that it should be employed with great caution in specially selected cases. The recommendation of Curschmann, to use a tube of such a form as to permit the entry and exit of air at will, is certainly worthy of consideration. It is more difficult to fulfil Curschmann's second condition—namely, the selection of suitable cases. Thus, it is often impossible to determine whether ulceration of a carcinoma of the colon has occurred and if there is danger of rupture. It is often exceedingly difficult to determine the nature of a stenosis. I believe that all stenoses due to ulceration or tumours constitute a contra-indication to insufflation with air. It is also advisable not to attempt this procedure in invaginations low down, as one can never determine with certainty how far the process has advanced (beginning gangrene). Careful insufflation may be done in simple volvulus of the sigmoid flexure when the diagnosis is certain.

3. Gastric Lavage in Intestinal Diseases

Gastric lavage in intestinal diseases comes up for consideration in stenosis or stricture of the bowel, above which fæcal matter has collected; also where fermenting stomach contents exercise an unfavourable influence upon the intestines. In the latter case, however, the retention of stomach contents in itself constitutes an indication for gastric lavage. Lavage may be advisable, though not necessary, in patients in whom an excessive production of acid or of mucus in the stomach causes an irritation of the mucous membrane of the small intestine, and frequently a resulting diarrhœa.

In general, lavage of the stomach is done for stenosis or stricture of the small intestine; but in obstruction or occlusion of the large intestine, according to numerous writers, it is often a life-saving procedure. The credit for having first tried and recommended gastric lavage in obstruction must be given to Kussmaul and his disciple Cahn[4]. From the observation of three cases, they showed that by means of this procedure cases of ileus with a bad prognosis could sometimes be cured. Soon after Senator-Hasenclever[5], Küster[6], Henoch[7], Ewald[8], Kauffmann[9], Kuhn[10], Curschmann[3], Pollak[11], and others reported successful cases, and at the present time we are justified in considering gastric lavage as a useful procedure, scarcely less important than opium.

As Nothnagel[12] quite properly observes, we must not expect too much from gastric lavage; indeed, that procedure at times may not only be ineffectual, but even harmful, since it can further diminish the already exhausted strength of the patients. The results from lavage would depend upon the nature of the obstruction and our ability or inability to overcome it by diminishing the pressure above. Since the nature of the obstruction in acute cases can usually not be determined, it can be readily understood that gastric lavage may be a life-saving procedure in one case, while in another case its effect will be an entirely negative one.

One point, however, should always be borne in mind: Gastric lavage should always be resorted to in the early stages of ileus. The more violent and fæcal the vomiting, the greater the indication for gastric lavage. For we recognise in fæcal or feculent vomiting a certain sign of a marked putrefaction of the stagnating contents above the site of strangulation, and only through the relief of this condition can we check the violent peristalsis of the bowel and the stagnation of its contents. As a rule, therefore, lavage is only indicated in those cases of ileus which have not progressed for more than twenty-four to forty-eight hours. In exceptional cases, the period when the general condition of the patient is a good one, lavage may be tried at a later period of the affection. Where there is already beginning heart failure, a miserable, rapid pulse, and possibly, too, impending death, the chances of accomplishing any benefit whatever with the procedure are very slim indeed. If a case such as that of Pollak * is now and then reported, in which, under most desperate conditions, a cure was effected, it only demonstrates the utmost extreme in which lavage may be of benefit. In private practice it is not always easy to resist the urgent wishes of those interested in the patient, who want every measure tried which can possibly save the patient, particularly if nothing can be lost and possibly much gained thereby. It will always be well, however, to point out the fact that such a mechanical procedure in the late stage of the affection may hasten the fatal termination of the case.

The condition which presents itself in chronic stenoses, and the indication for its relief, is much simpler. Of these stenoses, those of the small intestine, particularly of the duodenum (supra- and infrapapillary), call for washing out of the stomach. The conditions are so much like those of stenoses of the pylorus that they are to be treated just like the latter. We must not wait, therefore, with lavage until vomiting becomes incessant, but should begin as soon as the stomach contents give evidence of stasis (in infrapapillary stenosis, bile-stained or feculent).

It is quite evident, however, that only in rare cases can we secure permanent relief of the stenosis by lavage.

TECHNIC OF GASTRIC LAVAGE IN INTESTINAL OBSTRUCTION AND STENOSIS

This differs in a few points from ordinary methods of lavage. In the first place, the stomach must be washed out several times daily (two to five), and the washing must be continued each time until the stomach is entirely empty. The succeeding lavage must not be delayed until vomiting again occurs, but must be done as soon as fæcal matter again enters the stomach. This will be shown by renewed distention of the stomach, which has become less prominent immediately after the former lavage. The fact that only clear water flows away with the first washing must not deter us from fur-

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* Loc. cit. (Case IV).

ther lavage, particularly where the patient has previously vomited large quantities. I usually give a centigram of morphin subcutaneously before washing out the stomach. I have never had occasion to cocainize the pharynx. A hypodermic injection of caffein or camphor may be necessary previous to the lavage. Skilful technic (compression of the tube upon its withdrawal (Ewald)) will enable us always to avoid aspiration. The pulse is to be carefully watched in very weak individuals. The technic of gastric lavage in chronic stenoses differs in no wise from that of stenoses of the pylorus. Here, too, once a day, best in the morning, is sufficient; or in the evening, where rest is disturbed by the decomposition of the stagnating products.

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CHAPTER XII

MEDICINAL TREATMENT OF INTESTINAL DISEASES

1. Cathartics

By cathartics we understand those remedies which are capable of ridding the intestines of their contents within a short time. Since earliest times these have been divided into mild (*eccoprotica sive lenitiva*) and powerful cathartics (*drastica*). Between these classes there is a middle class, the so-called neutral salts, which belong to one or the other classes according to dosage employed and the individual effect obtained.

In spite of numerous investigations (confined for the most part, however, to animals), our knowledge of the action of cathartics is very limited. The effect of an individual cathartic varies in all probability with its chemical state. This accounts for the variety of action observed. Thus castor oil acts only after emulsification by the pancreatic juice and the bile; gamboge, elaterium, jalap, and scammony act similarly.

Calomel is changed in the small intestines; a portion of it is converted into corrosive sublimate. We do not know in what manner this substance acts; all mercurial salts cause increased catharsis. It is probable that the action of this drug begins in the upper part of the intestines, while other cathartics, such as senna, aloes, and colocynth, exert their main action upon the large intestine (Nasse, Radziejewski).

The action of the neutral salts appeared easiest of explanation. It was natural to suppose that their action was due to a transudation. As great an authority as Liebig supported this theory; he was followed by French investigators, chief among whom was Poisseuille. Later Aubert[1] opposed this theory, while Moreau[2] advocated it. Radziejewski[3] regards the influence upon peristalsis as the main principle in the action of cathartics. Brieger[4] showed that the action of laxatives is due to stimulation of peristalsis; neutral salts in addition cause an increased secretion of water and a larger secretion from the glands of the intestinal mucous membrane;

finally, that drastic cathartics in small doses act like laxatives, while in larger doses they cause an inflammatory exudate and a hypersecretion.

These unsatisfactory results include all that we know of the theory of the action of cathartics, and we must therefore place our dependence upon experience gained in daily practice.

In this connection my experience leads me to emphasize that every cathartic, or group of cathartics, has clinically a distinct action, and therefore distinct indications. For example, in acute constipation, castor oil, which acts rapidly and painlessly, is first to be considered; in acute indigestion, with or without bacteriological basis, calomel in large, rapidly repeated doses is useful. The use of the neutral salts or bitter waters is indicated in bilious individuals with hemorrhoids. Between these two are a whole series of other cathartics whose use is not especially indicated, but which are valuable only when varied, since most of them lose their action after a time. The physician should, however, have a knowledge of the main and secondary action of these remedies, and make his own indications.

Opium and belladonna occupy a special position among cathartics. We shall recur later to the effect of these substances upon the intestines. In this connection it need only be mentioned that they should be used where spastic contraction prevents the intestine from emptying itself. Lead colic is a typical example of this. In spastic affections of the intestine, flatulent colic, stenosis of the intestines, and in ileus, one can accomplish more by overcoming the spasmodic contraction of the bowels than by the use of cathartics themselves.

A large number of cathartics, once very much in favour, have now become obsolete. Among these substances we may mention agaricus, euphorbium, fructus colocynthidis, gamboge, hellebore, herba gratiolæ, leptandra virginica, metallic mercury, elaterium, croton oil, scammonium, and jalap tubers. A number of other remedies might with justice meet with the same fate. The following, on the contrary, are recommended if used under proper indications: castor oil, olive oil, calomel, magnesium salts, cascara sagrada, rhubarb preparations, tamarinds, podophyllin, precipitated sulphur, tartrate of soda, frangula bark, senna leaves.

With the exception of glycerin, subcutaneous and rectal administration of cathartics (aloes, etc.) has heretofore been but little employed in medicine, although the subcutaneous employment of physostigma for cathartic purposes has been well established in veterinary practice.

INDICATIONS FOR THE USE OF CATHARTICS

We must here differentiate between acute and chronic (habitual) constipation. The employment of cathartics in the first-named condition will depend principally upon the cause of the constipation—acute gastric catarrh, initial stage of acute infectious diseases, acute intestinal obstruction, typhlitis, appendicitis, invaginations, etc.

The employment of cathartics is indicated only where we can exclude inflammatory processes and displacements of the intestines. As long as there is any doubt, and there is no indication for active treatment of the acute constipation, one should limit himself to enemata, which can never do much harm. When there is a necessity for cathartics in acute constipation, a rapidly acting and certain cathartic should be used. If there are many stomach symptoms, calomel is to be recommended; where there are none such, castor oil is preferable. The various rhubarb preparations are valuable, but these lack the thorough action of the cathartics just named. No others need be considered in these cases.

In treating these conditions it should be remembered that cathartics should be stopped as soon as possible; it is therefore advisable not to give too small doses. If after one or more doses the constipation recurs (which is frequently the case), it is advisable to treat the patient according to the principles previously laid down—i. e., by dietetic measures.

In chronic (*habitual*) *constipation* the indication for the use of a cathartic, and the selection of the one to be used, depend upon the nature of the affection. We must distinguish between conditions in which the patency of the intestinal canal is diminished, whatever be the situation or the cause, and those in which the lumen of the bowel is unobstructed.

In the former class of cases, especially in stenoses of the intestine, cathartics are generally indispensable and symptomatically useful. Since a stasis of fæcal contents which is favourable to decomposition processes develops above the point of stenosis, cathartics are preferable to enemata, even when stenosis is situated low down. Enemata are generally without result.

The kind of cathartics to be used is a question of great importance; those only should be selected which do not cause painful spasmodic contraction and thus create a danger of perforation. These are the mildly acting preparations of rhubarb, compound licorice powder, magnesia usta, sodium phosphate, preparations of tamarind and cascara sagrada, tea of frangula bark, etc. Preparations of aloes, bitter waters, podophyllin, and the senna preparations are to be used with caution.

In the second class of cases—i. e., those in which the permeability of the bowel is preserved—the character of the disease determines the method of treatment to be pursued. In my opinion cathartics should be used only in desperate cases of constipation, and in old people in whom the normal reflex excitability of the intestinal muscle has disappeared. Here one should endeavour to use small doses of the milder aperients. Aided by a stimulation of intestinal activity by proper diet, this procedure will often, though not always, be successful.

The habitual use of cathartics in young persons is to be avoided. After a misuse of these for years, it is very difficult to succeed in bringing back normal peristalsis. I have already mentioned (page 149) that even in such cases one can sometimes obtain good results.

Contra-indications to the Use of Cathartics

In acute affections, in which the diagnosis of occlusion of the intestine is certain, the use of cathartics is contra-indicated. In the early stages of an acute intestinal affection, which later proves to be a case of intestinal obstruction, one can not blame the physician for having given a cathartic. In the later stages, where the symptoms of intestinal obstruction are clear, it is an error to give a cathartic. The same holds true for acute inflammatory affections of the type of an appendicitis. Our present knowledge of the nature of this disease (appendicitis) allows us to diagnosticate a typhlitis stercoralis in exceptional cases only, so that it is always advisable to keep in mind the possibility of a primary disease of the vermiform appendix. If, notwithstanding, there is reason for emptying the bowel, enemata are indicated (see page 180). When, in exceptional cases, there is a particular reason for one dose of a cathartic, castor oil is by all means the surest and best drug to use. It is far preferable to the saline cathartics, bitter waters, or rhubarb. In this connection we must mention that cathartics are also contra-indicated in the habitual constipation which so frequently follows appendicitis. In most cases the patients do well with proper feeding, or good results may be obtained from enemata, glycerin suppositories, etc.

Cathartics are contra-indicated in young people who have been constipated for a short time only. In children cathartics are absolutely contra-indicated, and their employment can not be too strongly condemned. In these cases continued and energetic care in feeding, combined, perhaps, with massage and gymnastics, gives good results. One should try dietetic measures for several weeks before resorting to the constant employment of cathartics. These drugs are contra-indicated in chronic gastric and intestinal catarrhs. As regards the former, I have gained the impression that cathartics have an unfavourable influence upon the secretion of the gastric juice. This seems to me to be the reason why we more frequently meet with insufficiency of gastric secretion in women than in men, though the latter are more apt to acquire gastric affections on account of their use of alcohol and nicotin.

Still more important is the contra-indication to the continued use of purgatives in chronic enteritis, affecting either the small or the large intestines. A careful observer who is accustomed to make frequent examinations of the fæces will doubtlessly have recognised that a slight enteritis of short duration, unnoticed by the patient, results not only from continual use of cathartics, but may also follow one or two doses of a mild laxative. This is much more marked where drastic cathartics are habitually employed, and I feel I am not exaggerating when I maintain that the more frequent occurrence of membranous enteritis in women is due to careless employment of laxatives. This is already shown from the fact that the mucous secretion ceases at once, and in most cases permanently, when intestinal functions are regulated by physiological laxatives. In the special part of this work I shall illustrate this by a series of histories.*

2. Antidiarrhœal Remedies

Since earliest times medicinal remedies have played an important rôle in the treatment of chronic diarrhœa. They are believed either to inhibit peristaltic action, thereby creating better conditions for the absorption of nutritive material from the intestinal canal, or restrict the production of intestinal secretion, or finally to cause neutralization of certain fermentative processes in the intestines. Antidiarrhœal drugs are furthermore employed to furnish a protective covering for intestinal ulcers, in so far as these are the cause of the diarrhœa and thereby to exert a curative effect upon the latter. Some remedies (calomel, tincture of rhubarb, belladonna, etc.) are laxative in large doses but binding in small ones. There are some drugs, too, which exert a beneficial influence in diarrhœa without our being able to explain their action.

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* von Noorden, in a very remarkable article upon membranous enteritis (Zeitschr. für prakt. Aerzte, 1898, No. 1) takes a stand which I maintained a number of years ago (cf. Deutsch. med. Wochenschr., 1883, S. 1000).

The correctness of the above statement of the action of drugs in diarrhœa may be disputed; certain it is, however, that acute and chronic diarrhœa frequently are cured when drugs are administered.

1. Remedies which inhibit peristalsis:

These include opium and its alkaloids (morphin, codein, narcein, and papaverin), as well as atropin and the belladonna preparations (particularly in small doses).

2. Remedies which act upon the intestinal mucous membrane (astringents):

These include tannic acid (tannin) and its preparations, tannigen (diacetyl tannic acid) and tannalbin (tannin albuminate). Among other remedies which contain tannic acid may be mentioned colombo, catechu, hæmatoxylon, cascarilla, ratania, oak bark, cotoin, and paracotoin. Nitrate of silver should also be classed with the intestinal astringents.

3. The antifermentative remedies include calomel, salicylate of bismuth and beta-naphthol bismuth (orphol), nosophen, paraform, tannoform, creosote, salol, beta-naphthol, etc.

4. The remedies which furnish a protective coating include carbonate and phosphate of calcium and similar chalk preparations (pulvis cretæ, talcum preparations, bismuth, etc.).

The Indications for and Use of Antidiarrhœal Remedies

Antidiarrhœal remedies are administered by mouth or as suppositories (opium, morphin, belladonna, etc.), or finally as rectal injections. Only rarely are they given subcutaneously. For rectal injection, tannin, nitrate of silver, aluminium (preferably the aceto-tartrate salt) or boracic acid are best. They are useful in diseases of the large intestine only, while the internal remedies are to be used in catarrhs of all parts of the bowel.

In some cases it is easy to determine which remedy to employ;

in others the difficulty of correctly interpreting the action of the remedies makes the selection of the proper one in a given case less easy.

Thus the use of antidiarrhœal remedies is clearly indicated in acute affections following exposure or errors in diet. The question arises in these cases: Should the remedy be given at once, or only after the bowels have been sufficiently emptied? In weak individuals the immediate administration is in general the better procedure; but where the general condition is very good, we should not be too eager to check a diarrhœa, particularly if this be accompanied by fever (beginning typhoid, infectious gastro-enteritis, etc.), or if there be an epidemic of cholera. On the contrary, it is much better to keep up the diarrhœa for several days by the administration of calomel—large doses at first, smaller ones later.

If none of these complications be present, we must give a rapidly acting and certain astringent remedy. Opium is the only one to be considered, and should be given in the form of the tincture, the powder, or the extract, combined, perhaps, with the tincture or the extract of belladonna. All other drugs, as well as any other combination with opium, are superfluous. As a rule, it is better to give the opium by the mouth than by the rectum; only where there is pronounced nausea or marked tenesmus should opium be given *per rectum*, and then in the form of suppositories combined, perhaps, with belladonna extract. Morphin, codein, papaverin, and other alkaloids of opium are inferior to the mother drug in their action. Not enough is known about subcutaneous injections of the watery extract to allow one to express an opinion as to their efficiency.

Opium, whenever indicated, should be given in effective doses, but continued for a short time only. I would strongly warn against small divided doses. The correct amount for an adult is twenty drops of the tincture two or three times a day, or two or three doses of the extract, each equal to 0.05 gram. With this and proper feeding an acute intestinal catarrh is generally cured. For eight to ten days the patient must be instructed in rules of diet (see section on Diet). The main use of opium is in *acute gastro-enteritis*; it is not a remedy for prolonged employment.

The other drugs above mentioned are used in a variety of ways and combinations in *subacute* and *chronic* diarrhœas, especially in those affecting the small and upper part of the large intestine. If we were to believe the praises which have been sung in medical literature about these drugs, especially during the last decade, the treatment of chronic intestinal catarrhs, even those of a tubercular variety, would seem to be as promising and simple as anything in all internal medicine. The critical physician would naturally doubt this statement if for no other reason than for the fact that new pharmaceutical remedies are being continually produced and recommended.

I have not seen any lasting influence upon chronic diarrhœas from any of the newer antidiarrhœal remedies. My experience in this respect has been a large one. My results in the treatment of these conditions on exclusively or almost exclusively dietetic principles (about which the most important facts have already been stated) have been just as good as when the newer remedies were used. The so-called antiseptic astringent remedies would be ideal if they exerted an influence upon the disease without simultaneous dietetic treatment, and if they were able to influence the mucous membrane in such a manner that the intestine would gradually become able to bear almost normal *régime* without difficulty and without the occurrence of diarrhœas. There is no real value in a constipating action *per se* of drugs.

It is true, I believe, that we can use antiseptic or astringent drugs as adjuvants to our dietetic and hygienic methods. The healing of an intestinal catarrh can be accomplished only by long-continued care of the intestines as has been described above, and in connection with long-continued absolute rest in bed. Whoever looks for impressive results will without doubt find great satisfaction from modern antiseptics and astringents; but whoever desires to make a permanent cure of a chronic diarrhœa can not place a very high value upon these remedies.

The indication for the application of astringent remedies *per rectum* is limited to chronic catarrhs of the large intestine. I have sometimes seen favourable results from tannin and nitrate of silver, but have obtained much better results from aceto tartrate of aluminium (one teaspoonful to the litre of water). It must be mentioned, however, that astringent remedies can not only cure catarrhs, but may cause or aggravate them. Great care in the administration and close observation of the reaction following such astringent injection is imperative. It is advisable to follow an enema of nitrate of silver by a neutralizing injection of salt solution.

CONTRA-INDICATIONS TO THE USE OF ANTIDIARRHŒAL REMEDIES

Above all, antidiarrhœal remedies are contra-indicated where the apparent symptoms are those of diarrhœa, but the real condition is one of constipation. These remedies should not be employed in the diarrhœa which accompanies intestinal stenosis. In carcinoma of the rectum there are frequent apparent diarrhœas which must be considered as decomposition products of stagnating intestinal contents. Cathartic measures would be indicated in such a case. It is also best to administer them to patients with alternating diarrhœa and constipation. In these it is advisable to wait several days before giving drugs, in order to come to some conclusion as to the nature of the intestinal disturbance. Several years ago I had occasion to observe a case in which the conditions were so complicated that careful observation for several weeks was necessary before it could be determined that the patient had chronic constipation, and not chronic diarrhœa. The patient, who was very ill, was cured when the constipation was overcome. Among the remedies contra-indicated in chronic diarrhœa, opium and calomel should be mentioned, as they are frequently wrongly used. Although I place a high value upon opium as a remedy in many diarrhœas, I always hesitate before using it in the chronic form. It is true, one gains the advantage of placing the bowels at rest for a short time, but meanwhile they become filled with putrefying decomposed material, which only reproduces a severer diarrhœa as soon as the opium effect has passed off. I allow the use of opium preparations as a palliative method only during travel, in society, etc., but even then with the greatest reserve. I need hardly mention that when there is a suspicion of ulcerations of the intestines calomel must not be used, or, if so, only under restrictions.

3. Sedative Remedies

Owing to the great importance of these in the therapeutics of intestinal affections, it is necessary to dwell briefly upon their significance and use. They include mainly opium and its principal alkaloid, morphin (which in this respect is superior to codein, papaverin, narcein, etc.), and belladonna with its alkaloid, atropin.

From experimental investigations very little has been learned regarding the manner in which opium and its alkaloids act. Nothnagel's[5] opinion that the intestinal paralysis results from stimulation of the inhibitory fibres of the splanchnic is directly opposed to that of Jacobj[6] and Pohl[7], who, after extensive researches, have come to the conclusion that the paralyzing effect of opium is brought about by diminished excitability of the intestinal wall. The latest investigations of Z. von Vámossy[8] tend to show that there is a benumbing of the brain centres which prevents transference in the brain of centripetal impulses of the pneumogastric to those centrifugal nerve paths that control intestinal movements.

We can not here give more than this brief reference. It shows sufficiently well, however, that we still have had no satisfactory explanation of the action of opium.

As regards belladonna and atropin, it is assumed that they cause paralyses of the intestinal wall, and thus relieve abnormal contractions of the intestinal coils.

We know more, however, about the clinical application of narcotics. The following are the indications for their use: To allay inflammation, to relieve painful contractions, to arrest increased intestinal peristalsis accompanied by profuse evacuations of contents, and, finally, to secure rest of the entire intestines, so as to permit of a correction of intestinal knottings, invaginations, incarcerations, and volvuli.

Appendicitis furnishes the best example for the use of opium in inflammatory processes. We should, however, make a careful distinction between the various stages of this affection. In appendicitis, opium is only indicated when symptoms of irritation, fever, pain, and meteorism are present, and the patient's condition is still satisfactory. If these symptoms disappear after repeated large doses of opium (e. g., tinct. opii gtt. xx every three hours, or extr. opii 0.03 gm. t. i. d.), its further use, though not harmful, is no longer necessary. If the opium is continued the dosage should be reduced one half. If symptoms of irritation have been present for three or four days, opium is contra-indicated, for then there develop intestinal paralyses, which are overcome only with the greatest difficulty.

If symptoms of irritation continue and those of collapse (rapid, compressible pulse, general depression, etc.) begin to appear, opium must at once be stopped. This is the stage in which opium, as surgeons very correctly claim, masks the disease and gives rise to false hopes.

A similar rule applies to acute intestinal occlusions. Here also opium is indicated in the early stage with symptoms of reaction.

If opium does not then yield the desired results, a curative action can no longer be expected from it; it can only induce euthanasia. The same objections against the giving of large doses apply as in appendicitis. Undoubtedly large doses of opium temporarily relieve the seriousness of the condition, but actual benefit can no more be expected than from an infusion of digitalis in a cardiac case with impending paralysis. The pulse and general condition should guide us in the management of acute intestinal obstruction. If these are good and the features "composed" (von Leyden), opiates will at least do no harm; if these are not good, then opium can only help seal the patient's fate.

A few words regarding the treatment of enteralgias, intestinal colic, peristaltic restlessness, and acute diarrhœas. Whatever may be the etiology of these conditions, narcotics will always be of benefit, for they quiet the excessive peristalsis, relieve pain, and thus enable solid contents to pass through the intestinal canal, and fluid contents to become more solid.

In a given case it is by no means immaterial whether we administer opium or one of its alkaloids (particularly morphin) or belladonna (or atropin). This has been sufficiently shown by clinical experience. Opium is far superior to any of its alkaloids in quieting the intestines or in arresting peristalsis. Morphin has only the analgesic property of opium. In any case in which we desire a sedative effect upon the intestines, as well as a slowing or an arrest of peristalsis, opium will be found superior to all other narcotics.

On the other hand, extract of belladonna in doses of 0.01 to 0.03 gram, and its alkaloid, atropin, in doses of from 0.005 to 0.001 gram, are indicated in all colicky and painful crises. Like the opiates, they may in such cases even have a laxative action. In general, their action is weaker than that of the opiates; their long-continued employment is always attended by danger of poisoning. Opiates are frequently combined with belladonna.

4. Remedies for Flatulence

Since early times a large number of remedies have had the reputation of allaying flatulency, either through combination with the gases or excitation of the intestinal motility, or through other unknown ways. As regards the power to combine with gases, the magnesia salts, especially magnesia usta, play an important part. It is well known that outside of the body magnesia usta can absorb a large quantity of carbonic-acid gas (1 gram absorbs about 1,100 cubic centimetres). To what extent this substance can absorb gas in the gastro-intestinal canal is still a matter of question. In the stomach, magnesium chlorid is formed by combination with hydrochloric acid; in the intestines only the residue of the magnesia usta can be converted into magnesium carbonate. It is very probable, too, that this formation of carbonate of magnesia does occur in the intestinal canal, for, after the ingestion of magnesia, patients frequently have borborygmi and expel much gas. To a certain extent, however, the laxative action of the magnesia may cause such symptoms.

The second group of remedies—i. e., those which excite intestinal motility—is said to act by increasing the muscle tone. It includes nux vomica and its preparations (the tincture, the fluid extract, and its alkaloid, strychnin), as well as extract of physostigma. We will speak of these more in detail in the next division.

Very little is known of the action of the so-called carminatives. But here, as everywhere in science, experience is the best guide, and it has shown us that in flatulency very great benefit is often derived from the use of carminatives.

The carminative remedies include various so-called carminative teas, such as valerian, fennel, peppermint, caraway, anise, thyme, and the oils obtained from these substances, the most frequently employed being oils of menthol, caraway, and fennel. These remedies very probably have mild antiseptic properties, such as the oil of menthol has been shown to possess. Based on this antiseptic property, menthol (0.1 to 0.2 in gelatin capsules) has been recommended for the relief of flatulency.* The following sums up the latest ideas regarding the relative value of the various preparations. With teas made from the fruit or leaves there is the additional effect derived from the heat. We know from experience that heat when applied externally, or hot solutions taken internally, exert a beneficial action on acute colic and flatulency. The oleosaccharates and the oils are more in place in the chronic forms of flatulency. The oils are to be given singly or combined, in doses of 3 to 5 drops thrice daily, and the oleosaccharates in amounts varying from very minute quantities to a teaspoonful, also thrice daily. The combination of magnesia usta with the so-called oleosaccharates is very effectual where constipation exists. For example:

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* To insure solution it is necessary to drink an alcoholic mixture (cognac or wine) after the dose is taken.

℞ Magn. ustæ	15.0
Olæosacch. menthæ	5.0

M. Pulv. Sig.: A quarter to a teaspoonful three times a day.

In a number of cases of chronic flatulency, pulverized caroway fruit in tablet form, in doses of from 0.5 to 1 gram, has proved very serviceable to me.

Charcoal in the form of freshly burnt animal or wood charcoal is still very frequently given as a gas-absorbing remedy. In France, especially, the "pastilles de Bellocq," which consist mainly of charcoal, are very generally used. We can hardly expect any benefit from charcoal, however, since it only absorbs gases while it is in a dry state.

5. Tonic Remedies

Are there any remedies which may rightly be called intestinal tonics? Concerning this subject little has been learned from animal experiments. Although there are no exact indications for their use, and no convincing results have been obtained, drugs are often employed for this purpose. They include the numerous bitters which, according to the investigations made by Ramm[9] and Kobert, stimulate intestinal as well as gastric peristalsis; furthermore, the preparations of nux vomica are also given (tincture of nux vomica, gtt. x–xv, extract of nux vomica 0.01 to 0.03, or nitrate of strychnin 0.001 per dose, preferably subcutaneously). The other bitters (tinct. amara, gentian, creosote, etc.) also enjoy considerable reputation as intestinal tonics. Besides these remedies, I have frequently obtained good results in painful flatulency from the use of the extract of calabar bean. Attention was first directed to this drug by Subbotin[10], and later by S. Schaefer[11].

℞ Extr. physostigmæ	0.05
Glycerin ad	10.00

M. Sig.: Ten drops three times a day.

Or the same remedy may be given in pill form in doses of 0.005 gram. It has no laxative action. I have never seen any injurious effect result from the extract of calabar bean, and I am convinced that in atony of the intestines this drug is as worthy a trial as nux vomica.

6. Intestinal Antiseptics

Owing to the numerous investigations of Bouchard and Dujardin-Beaumetz and their scholars, the idea that it is possible to disinfect the intestinal canal in fermentative and putrefactive conditions has been widely accepted, especially in France. These investigators have evolved a method of treatment which has been named "antisepsie intestinale." Although it had many enthusiastic followers in Germany, this movement has been reluctantly taken up. Recently a marked reversal of opinion has taken place in France[12], for the lack of results spoke too strongly against the dogmatic certainty with which the new teaching had been proclaimed.

Regarded critically, the idea of intestinal antisepsis really rests upon a very weak foundation; for we have little knowledge concerning the manner in which putrefactive products originate in the intestines, as well as the factors which increase or diminish them; and, furthermore, we do not know which of these products are absorbed and which are not, nor how the various foodstuffs influence them. Through the investigations of Rovighi[13], Schmitz[14], Winternitz[15], and Albu[16] one fact has been established—that milk and its products (kefir and pot cheese) cause a diminution in the ethereal sulphuric acids in the urine. Furthermore, it must be remembered that inhibition of normal intestinal motility results in an increase of putrefactive products. If we compare the conditions existing in the stomach with those in the intestines—more complex, it is true—we will have a basis upon which to study the question of intestinal disinfection. We know to-day that the best way in which to disinfect the stomach is to restore to it its normal motility. Therefore it must be recognised that by the mechanical evacuation of the stomach, numerous fermentative products and their causes are removed, and by this means a more favourable condition is created. Objectively this is noticeable in a demonstrable increase in the strength of the distended organ. If the normal motility be lost, permanent disinfection of the stomach, filled as it is with microorganisms, is impossible.

The knowledge gained from these experiences may be applied to the far more complicated conditions in the intestines; here also the best antiseptic remedy is the restoration of normal peristalsis. Therefore Albu[17] is perfectly correct if, in his numerous publications upon this subject, he regards catharsis as the main weapon with which to combat intestinal putrefaction.

A second scarcely less important means of mechanically combatting the causes and results of intestinal putrefaction is careful, systematic intestinal lavage. It is well known, and we, too, have repeatedly pointed out, that decomposition takes place mainly in the large intestine; the utility of careful and repeated intestinal lavage is therefore very apparent. By this procedure we do not attempt to sterilize the intestine, nor do we thereby make it impossible for new microbes to develop, but we obtain at least a relatively clean condition of the organ. Intestinal lavage is beneficial also from another standpoint: a portion of the water is absorbed, causes an increase in the excretion of urine, and thus, as Sahli was the first to show, numerous products of decomposition are washed out of the system.

For intestinal lavage a number of antifermentative substances may be added to the water: salicylic acid, boracic acid, resorcin, creolin, lysol, creosote, ichthyol, tannic acid, and perhaps also formaldehyde and its preparations (amyloform, dextroform, paraform, tannoform, etc.). Very little has been published regarding the last-named preparations. Undoubtedly disinfection of the intestine is best accomplished by means of lavage. As regards the technic, the only point to be remembered is that the irrigation must be continued until the water returns clear. As in gastric lavage, so after apparent cleansing of the intestines, a change in the position of the body will often cause fresh fæcal masses to appear and be washed away.

In spite of the numerous remedies which have been recommended and tried within the last decade, no important results have been obtained for the disinfection of the intestinal canal by way of the stomach. Nevertheless, it seems to me that Albu goes too far when he says that internal antisepsis, in the broadest sense of the word, is an unsolvable problem. The solution certainly will not be found in a chemical agent, but rather in a proper selection and preparation of the ingested food. In the future, therefore, it should be our duty to study more closely than heretofore the effects of the various foodstuffs upon decomposition processes in normal and abnormal conditions. We must cease to place any value upon methods for the estimation of proteid decomposition from the amount of ethereal sulphuric acid found, or upon the relation between preformed and total sulphuric acid. This method has led to the utmost confusion.

After what has just been said, it will be unnecessary to enumerate and discuss the numerous intestinal antiseptics which have been recommended. A few of them, however (salol, orphol, nosophen, bismuth salicylate, etc.), have, as already mentioned, a distinct value in the treatment of chronic diarrhœa. This subject will be treated of in detail in the chapter on Diarrhœa, in the second part of this work.

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PART II

SPECIAL DIVISION

CHAPTER XIII

ACUTE AND CHRONIC INTESTINAL CATARRH

(*Enteritis acuta et chronica*)

1. Acute Intestinal Catarrh

Preliminary Remarks.—Acute intestinal catarrh owes its importance not so much to the gravity of its symptoms as to the frequency of its occurrence. It affects persons at all ages, more particularly, and to a very dangerous degree, children during the first two years of life. There is no difference as regards sex. Certain periodic variations, dependent upon atmospheric and climatic influences, exist.

The causes of acute enteritis, as of acute catarrhal gastritis, are very numerous. Its various forms, however, may, on the whole, be traced to the following four primary causes:

(1) To infection (infectious catarrhs).
(2) To injurious substances in the ingested food (alimentary or ingestive catarrhs).
(3) To cold (refrigeration catarrhs).
(4) To the action of medicines or poisons (medicinal or toxic catarrhs).

Infectious catarrhs, according to our present knowledge, are due to bacteria or to their metabolic products. The sole primary manifestation of the disease may here be limited to the intestinal canal, or the intestinal catarrh may form the chief or accompanying symptom, or be a complication of other acute infectious diseases (typhoid, cholera nostras et asiatica, pneumonia, malaria, anthrax, influenza, sepsis, etc.).

We are here concerned only with the first-named variety. *A priori*, it seems probable that in infectious catarrhs a specific bacterial flora does not exist, and, despite manifold studies in that direction, the demonstration of a specific micro-organism as the cause of intestinal catarrh has been only partially successful. In this connection we would mention Gaffky's[1] demonstration of the bacillus enteritidis (probably identical with the bacterium coli) in a severe case of infectious intestinal catarrh. An important though not necessarily the sole factor in the etiology of acute enteritis is the bacterium coli.

Alimentary enteritis is the most common form met with. Decomposed food, or else wholesome nourishment ingested in improper combination or in excessive quantities or of unsuitable physical character, may give rise to violent intestinal disturbances, which assume the character of an acute intestinal catarrh. Among the foods which most frequently excite intestinal catarrh are water, ice, milk and its products (butter, cheese, whey), meat and its derivatives, sausages, fish, fruit, etc. It has been justly assumed that poisonous organic bases (ptomains) play an essential part in these cases. Some of these have already been determined (Vaughan's tyrotoxicon, Firth's lactotoxin, and the ptomains muscarin, guanidin, methylguanidin, dimethylamin, methylamin, æthylamin, betalin, mytilotoxin, etc., isolated by Brieger). In consequence of improper combination or excessive ingestion of otherwise wholesome food, injurious products of fermentation (lactic, butyric, propionic, acetic, caproic, formic, succinic acids, etc.) may be formed, which, innocuous in ordinary quantities, may under the above-named condition cause violent irritation of the intestinal canal.

Undoubtedly we also meet with diarrhœas due to exposure to cold, in which, perhaps owing to reflex vaso-motor influences, a marked stimulation of the excito-motor nerves is produced. When the irritation is violent or the patient careless, an acute intestinal catarrh may follow.

Finally, intestinal catarrh may result from the action of drugs, either through the use of abnormally large single doses or from the long-continued use of small amounts. Among these are the mercurial preparations (calomel, corrosive sublimate), arsenic, antimony; the emetics—tartar emetic, ipecacuanha, sulphate of copper, apomorphin; the strong drastic purgatives (croton oil, colocynth, senna, jalap, gamboge, etc.). Many other medicinal agents, administered in unsuitable manner or dosage, may produce intestinal catarrh; those named above, however, are the most frequent offenders.

As rare causes of acute enteritis we may mention traumatism, the presence of numerous entozoa, and acute coprostasis.

Symptomatology and Diagnosis

The following description relates solely to acute primary intestinal catarrh; all secondary varieties of enteritis—e. g., those that occur in the course of typhoid fever, cholera, tuberculosis, malaria, pneumonia, etc., carcinoma of the intestines, congestion of the portal circulation, intestinal ulcerations, intestinal strictures, entozoa, etc.—will not here be considered, because foreign to the present discussion. As regards the intensity and extension of the process, the symptomatology of acute enteritis presents a variety of graduated stages, so that it is difficult to faithfully describe all grades and forms of the disease. The following description applies to a moderately severe case of enterocolitis. Of especial importance are:

1. The commencement of the process.
2. The gastro-intestinal disturbances.
3. The patient's general condition.
4. The course of the disease.

Certain other symptoms, which will be discussed below, may complete the clinical picture.

As a rule, the symptoms appear suddenly and without prodromata, almost immediately following the exciting cause. Tormenting, dragging, and boring pains are felt in the abdomen, usually accompanied by general discomfort, nausea, and occasionally even by attempts at vomiting. Diarrhœa soon develops. The number and character of the evacuations vary according to the localization, intensity, and etiology of the disease. Thus in very mild cases there may be two to three, in severe cases fifteen to twenty or more passages. The first evacuations may still be solid or pasty, but the more numerous they become the more fluid and odourless will they be. Should the catarrh involve the upper portions of the small intestine, we may recognize its site from the greenish-yellow colour of the dejecta and from the presence of unchanged bile pigments (Gmelin's reaction). If the catarrh is limited to the large intestine this test can no longer be employed, since the bilirubin has already been converted into urobilin.

All the stools of acute enteritis contain an admixture of *mucus*, partly in the form of exceedingly small, barely visible gelatinous particles or minute specks intimately mixed with the stool, partly in the form of isolated masses which consist of small or large shreds. Besides mucus, blood is not infrequently present, partly as small streaks usually adherent to the particles of mucus, partly in the form of larger bright red or coagulated masses, also generally mixed with the latter. In my experience copious hemorrhage is very rare in acute enteritis. In the majority of cases of uncomplicated enteritis, unlike those of dysentery, *pus* is not present in the evacuations. Owing to the active gas formation within the intestines, the fæces may present a marked frothy appearance. Besides mucus, the microscopical picture reveals red and white blood-corpuscles, and frequently partly fresh but mostly disintegrated epithelium. We also find various kinds of food remnants, particles of casein, muscle fibres, starch granules, remnants of cellulose, and other accidental constituents of food.

Gastric digestion is frequently interfered with along with the intestinal disturbance. The appetite is generally absent, direct aversion to food is occasionally present; in other cases, gastric disturbances—nausea, vomiting, epigastric pressure—are so prominent that the case resembles one of acute gastritis. The tongue is usually coated; a disgusting fetor is present; thirst is frequently tormenting. There is an inverse relation between the number of stools and the thirst on the one hand, and the urinary secretion on the other.

The general condition depends essentially on the severity of the enteritis and the number of evacuations. Only in very few cases does it remain undisturbed; in the greater number of cases the patient complains of considerable lassitude, which in children, in the aged, or in otherwise weakened individuals not infrequently develops into symptoms of grave prostration. Under these circumstances, a condition of acute hydrocephalus, as described by Marshall Hall, may supervene, due to the copious intestinal discharges and the resulting cerebral anæmia.

In the vast majority of cases, especially in adults, the disease runs a favourable course and is of relatively short duration. Nevertheless, we occasionally observe, especially in colitis, quite severe and protracted forms, in which convalescence is long delayed. Other cases are characterized by a persistent vulnerability of the intestine, so that under predisposing conditions relapses frequently occur; the process does not go on to perfect recovery, a chronic enteritis gradually develops, or a more or less intractable atony of the large intestine, accompanied by constipation, remains.

Certain complicating conditions may be added to this typical picture. The disease, which usually runs an afebrile course, may be accompanied by more or less severe fever, so as to awaken the suspicion of a beginning typhoid, the more so as enlargement of the spleen is occasionally observed. The urine is generally concentrated and dark; in rare instances anuria may develop. It has been observed by various authors, first by Kjellberg[2] and soon after by Hermann[3] and Mühlhauser[4], that albuminuria may occur in cholera diarrhœas; and Kobler[5] has recently reported two cases of cholera nostras, in which for a long time there was a marked diminution of urine with albumin and large quantities of hyaline and epithelial casts. In one case granular casts and renal epithelium could also be demonstrated. Even in the milder forms of diarrhœa, as first shown by Fischl[6] and corroborated by Stiller[7], temporary albuminuria and cylindruria may occur. I recently made the same observation in an elderly patient, in whom on the second day of an acute gastro-enteritis I found an appreciable amount of albumin in his urine (without casts). With the improvement of the intestinal catarrh the albuminuria soon disappeared.

In exceptional cases in children, according to Turner[8], fatal Bright's disease may follow an acute gastro-enteritis.

Several authors (Strümpell[9], Fleischer[10]) report the occurrence of acute pains in the joints and muscles, as well as tumefaction of the joints, as a complication of acute enteritis. Herpes labialis has also been observed in infectious intestinal catarrh.

As a rule, the diagnosis of an acute enteritis is not difficult, especially where a positive etiological factor (errors in diet, cold, etc.) points to the origin of the disease. The diagnosis is based on the acute onset, the characteristic gastro-intestinal disturbances, and the course of the disease. Compared with these data, the objective abdominal symptoms are of subordinate importance. Inspection may reveal a slight distention, although this may be absent; in greatly emaciated persons, isolated spasmodic intestinal contractions, especially of the small bowel, are occasionally observed. If the colon is especially affected, palpation elicits some tenderness on pressure, particularly pronounced in the region of the descending colon and the sigmoid flexure, and thus may indicate the limits of the inflammation. In diffuse enteritis, the whole abdomen, especially the circumbilical portion, is the seat of more or less decided tenderness. No practical data are obtained by percussion. Auscultation enables us to hear loud borborygmi and other intestinal sounds even at a distance, and better still when the ear is directly applied to the abdominal wall. If we except the gastro-duodenal catarrh accompanied by jaundice which will be considered later, the localization is more difficult than the diagnosis of the diseased process. In my opinion, the only determination of some practical value is whether the seat of the catarrh is in the large or small intestines. The absence of large appreciable masses of mucus and blood from the stools and the presence of unconverted bile pigment is diagnostic of catarrh of the small intestine, while, conversely, the presence of a larger quantity of mucus and blood in the fæces argues in favour of disease of the colon. If we go still further, and attempt to localize the catarrh in the different intestinal segments, we are no longer treading upon scientific ground.

In view of the symptoms above mentioned we will meet with diagnostic difficulties only in exceptionally complicated cases. Thus, for example, during the first days of febrile gastro-enteritis the differentiation from typhoid, as already stated, may be exceedingly difficult, and may only become possible after careful observation of the course of the disease. This same difficulty confronts us in cholera asiatica, particularly when that disease is epidemic. In both instances the bacteriological and the serum examination (Widal's reaction) may materially assist the formation of a diagnosis.

The diagnosis of duodenal catarrh conforms completely with that of acute and subacute gastric catarrh, and only becomes possible with the onset of catarrhal jaundice. Its symptom-complex is generally so pronounced that a special description seems superfluous.

In my opinion, the differential diagnosis between acute colitis and the milder forms of dysentery is exceedingly difficult; I believe that we are as yet unable to satisfactorily discriminate between these two diseases.* In pronounced cases, and particularly during epidemics, the diagnosis is probably less difficult. In tropical dysentery, the presence of the amœba coli (Loesch) in the fæces and in the pus of the liver abscesses is of diagnostic importance.

The *treatment* of acute intestinal catarrh is based upon the following principles:

1. The removal of noxious material from the intestinal canal (indicatio causalis).
2. The greatest possible protection of the gastro-intestinal canal (indicatio symptomatica).

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* That this same difficulty has been encountered by other authors is obvious from the fact that in literature we not infrequently meet with the designation "dysenteroid intestinal catarrh."

We are not always able to carry out the *first* rule, since only in a certain proportion of cases are the noxious materials of a substantial nature. Where this indication can be fulfilled, recovery, as a rule, immediately follows the removal of the undigested masses. Frequently this result is also brought about by the products of decomposition themselves, which accumulate in the intestinal canal and excite increased peristalsis. Further treatment is then hardly necessary. Where, however, in spite of the obvious presence of noxious material, the evacuations are arrested or insufficient, the removal of this material by a suitable purge is of primary importance. For this purpose, calomel (0.2 to 0.3 grams, every three hours) [until the desired effect is obtained] or castor oil (in doses of one to two tablespoonfuls) is preferable.

Since the early days of medicine it has been customary to give calomel preferably in "infectious" catarrhs, and castor oil in cases due simply to errors in diet; still, the superiority of the one over the other is by no means established. In my opinion, both act as disinfectants by rapidly and thoroughly emptying the bowels.

The *second* indication—protection of the gastro-intestinal canal—must be considered after the first has been fulfilled, or where, owing to the absence of recognisable etiological factors, this is unattainable. This second indication is principally fulfilled by complete rest in bed for several days, by constant and uniform application to the abdomen of heat in the shape of warm fomentations, and most especially by a proper diet. For the first two or three days the diet should consist of fluids only. All liquids, however, are not suitable; for example, milk is contra-indicated in all forms of acute enteritis. Cold drinks as well as carbonated waters should also be avoided, excepting when severe vomiting is also present for experience has shown that ice and iced drinks tend to check the nausea. In acute cases the diet should therefore be limited to the following: watery gruels, thin bouillon without salt or spices, tea or cocoa made with water alone (no sugar, but in its stead saccharin), claret with or without water, egg albumen, decoctions of arrowroot and hygiama gruels, etc. Carminative teas are also frequently useful—e. g., peppermint, valerian, fennel, anise seed, thyme, caraway, marsh mallow, etc.

Only after the intestine has become *absolutely quiescent*, and tenderness to pressure has disappeared, may we increase the diet by adding one or more softened zwiebacks, next permitting the lightest forms of meat and fish, with some boiled rice or mashed potatoes, gradually approaching the ordinary diet. The patient should be instructed to carefully abstain from excesses in food and drink for a number of weeks, to avoid all indigestible, fatty, tough foods, and also those rich in cellulose, for, as previously stated, the intestine is very vulnerable for a long time.

These measures are all that are required for the cure of a large number of cases. It will only exceptionally be found necessary to control excessive diarrhœa, or, on the other hand, to combat a reactive constipation or to stimulate a failing appetite. The first is accomplished by the administration of opiates in the form of tincture of opium (10 to 15 drops, repeated one to four times) or the extract of opium (0.03, repeated two or three times a day), internally or in suppositories. I regard the continued administration of opiates not only as unnecessary but even as injurious. *When, as frequently happens, constipation occurs after the diarrhœa has subsided, this must not be treated by purgatives, but only by suitable enemata of oil or of soapsuds.* The appetite generally returns in due course of time. It may be stimulated by the well-known stomachics (the fluid extract or decoction of condurango, the tincture of nux vomica, the compound tincture of cinchona, etc.).

In conditions of grave collapse we may endeavour to arouse the heart's action by stimulants administered internally (e. g., wine, cognac, ether) or subcutaneously (e. g., oil of camphor, citrate of caffein).

2. Chronic Intestinal Catarrh

Preliminary Remarks.—Chronic enteritis may result from acute intestinal catarrhs in which recovery has taken place only incompletely or not at all, or from long-continued and various kinds of injuries of the intestinal canal. Among the injurious agents which conduce to chronic intestinal catarrh, those which directly affect the gastrointestinal mucous membrane are the most important and the most frequent. These include improper food, frequent colds, abuse of purgatives, constipation, etc. *The primary forms of enteritis* are caused in this manner. As distinguished from these, the *secondary* forms are those which develop either in connection with gastric diseases (e. g., catarrhs, carcinoma, fermentation, etc.) or from intestinal diseases (tumours, ulcerations, stenoses, adhesions, displacements, etc.). The intestinal catarrh is often overshadowed by the other predominant symptoms, or it may be so prominent that the etiological factor is only of secondary clinical importance. Nothnagel[11] correctly remarks that a congestive enteritis, in the true sense of the word, does not exist; at the most, we may concede that hyperæmia of the intestinal tract creates conditions favourable to the development of chronic catarrhs. As a matter of fact, I believe they occur very rarely as secondary catarrhs dependent upon the main disease; and when they do occur, who can prove that they have developed in this way, and are not accidental complications?

Clinical observation combined with post-mortem examination teaches us that we must distinguish between different grades and forms of enteritis, and that these affect various intestinal segments. For the better understanding of this I preface my remarks with a short description of the pathologico-anatomical changes that are found.

On macroscopical examination of such a diseased bowel the attention is immediately arrested by a brown or black pigmentation of the mucous membrane, which particularly affects the region of the villi and that of the follicles. Peyer's patches may also present such discolourations, caused by old hemorrhages. The histological changes involve chiefly the mucosa; they may also involve the submucosa, or the muscularis. The changes may consist of a cellular infiltration of the interstitial connective tissue (leucocyte infiltration), from which a genuine hypertrophy of the connective tissue may develop and this may occur to such a degree that the intestinal lumen becomes narrowed for quite some distance. In other cases there is a polypoid proliferation of the tissues. In still other instances there are cystic dilatations (enteritis chronica cystica), produced by strangulation of conglomerations of glands. Usually the cysts are small and hardly visible to the naked eye, but they may attain the size of a pin's head or a lentil. Their contents consist of a peculiar slimy, viscid fluid, whose clinical nature has not as yet been positively determined. In contrast to these plastic inflammatory processes a degenerative form can also be distinguished. This affects the glands rather than the connective tissue, destruction and atrophy of the glands gradually occurring as a result of the marked epithelial desquamation. At the same time the other layers of the mucous membrane generally lose their powers of resistance. The mucosa becomes thin and lamellated, the villi atrophied, so that on section we are only able to recognise isolated remnants projecting from the general level of the mucous membrane. Besides these changes, the process especially affects the intestinal follicles, which are swollen at first and are surrounded by a hyperæmic zone; later they rupture, and thus give rise to so-called follicular (lenticular) ulcers. In addition to these last, other erosions result from desquamation and loss of epithelium. At first they are superficial, but through their confluence and deeper extension true catarrhal ulcerations may gradually be formed.

Pathological anatomy not only distinguishes between the variety, but also between the situations of morbid processes, and hence speaks of a *duodenitis, jejunitis, ileitis, colitis, and proctitis.* Clinically, these fine distinctions can not be made, and we must be content with a division into *catarrh of the small* and *catarrh of the large intestines.*

Since catarrhs of the small and of the large intestines, however, frequently coexist, we must also consider this combination clinically. In an anatomical sense the one will naturally preponderate over the other, while the one intestinal division is extensively diseased, only a small portion of the other will be affected. In the present state of our knowledge these fine gradations can scarcely be made, and in view of the great number of variations met with they are of little practical value. The clinical diagnosis, therefore, will have to deal mainly with the following two questions:

1. Is an intestinal catarrh really present?
2. Does it involve the small or large intestine alone, or both?

In the following we will first outline the general symptoms of intestinal catarrh, and then will consider its localization.

GENERAL SYMPTOMATOLOGY AND DIAGNOSIS OF CHRONIC INTESTINAL CATARRH

In chronic intestinal catarrh the clinical picture is mainly formed by the subjective symptoms, or by objective abdominal manifestations—especially, marked variations in the number and character of the stools.

The subjective symptoms vary greatly in degree. They may be absent, or may only manifest themselves by disturbances of defecation; they may be only just felt, or may constitute the main complaint of the patient; they are constant or periodical, may disappear with or without the objective symptoms, or *vice versa*; they may persist, notwithstanding the cessation of the characteristic intestinal symptoms. Daily observation furnishes numerous examples of all the variations. We will have to refrain from describing them all, since an exhaustive description would not be possible. We shall, however, describe a few forms of chronic enteritis that are of practical importance.

In typical cases of chronic intestinal catarrh the symptoms consist in feelings of discomfort or pressure, pain or soreness in the abdomen, combined with intestinal rumbling and hyperperistalsis, followed usually by copious alvine evacuations. In marked cases these symptoms may be constant, or they may occur only in paroxysms—e. g., following a slight cold, excessive mental or bodily exertion, dietetic errors, mental excitement, etc. The patient at the same time has a sense of intense discomfort, of bodily and mental depression, to which are sometimes added salivation, retching, or actual vomiting. Where the intestinal catarrh is situated very far down, painful tenesmus is also present. In *atypical* or less marked cases the symptoms are confined to uncomfortable painful sensations which involve the whole abdomen, or else are limited to certain areas—e. g., the umbilical region or portions of the large intestine. When gas or fæces are passed these sensations may rapidly disappear. I would call particular attention to the frequent occurrence of this form of intestinal catarrh, for I am convinced it is only rarely recognised, since it may be accompanied by apparently normal fæcal evacuations. By careful observation and questioning of the patient, but especially from an examination of the dejecta, we may convince ourselves that these patients are affected with a slight or only partially healed intestinal catarrh. This will be again referred to in discussing the diagnosis. In another form of intestinal catarrh—the coprostatic—the subjective symptoms are entirely different. The above-mentioned symptoms are either entirely absent, or they appear as periodic intestinal colics, with spastic obstipation, or else in the form of a constant or very frequent feeling of pressure in the dependent portions of the intestines, or even as meteorism accompanied by more or less marked flatulency.

Between these two extremes we meet with cases in which the first-described combine in the most diverse manner with the last-described varieties.

The general condition of patients suffering from chronic enteritis varies very markedly. As a rule, it is only slightly altered from the normal in constipating enteritis, but very much more so in that form accompanied by copious diarrhœa, especially where the upper portions of the intestine are principally affected. Occasionally, however, we meet with chronic diarrhœas in which there is no deterioration of the general health, while, on the other hand, it is not rare to find cases of constipating enteritis in which the bodily weight and general condition have been gravely affected. This variable behaviour can not in all cases be explained alike. In a great number the psychic factor—i. e., the general depression—no doubt exerts an unfavourable influence upon the ingestion of food; in others, again, the loss of fluids is the main cause; in still others an irregular mode of life, errors of diet, excessive bodily or mental work, will cause a loss of strength and weight. The longer the disease exists and the more frequently curative measures fail to bring relief, the sooner will the patient present a certain neurasthenic tendency, which may even to the most experienced physician obscure the true cause of the malady.

The *objective examination* is divided into that of the abdomen and that of the dejecta. In the first, *palpation* alone possesses positive value. Percussion and auscultation may perhaps demonstrate the existence of meteorism, of accumulated fæces, of abnormally increased intestinal sounds, but they do not of themselves permit of any conclusions as to the existence, location, or extent of the intestinal catarrh. On the other hand, palpation may sometimes be of value in determining a tenderness of certain intestinal areas, thus, as will be later explained, indicating the seat of the intestinal catarrh. The evacuations vary very much, according to the nature and seat of the disease. The movements may be tardy, or may occur at regular intervals; they may occur too frequently or in excessive amount, or after several days of constipation there may suddenly be a single or repeated diarrhœal movement; or, finally, after, a period of days or weeks of perfect health, suddenly, and often without any recognisable cause, a normal stool may assume a diarrhœal character.

Of still greater importance are the changes in the composition of the fæces, as well as the presence of abnormal admixtures, particularly the constant occurrence of mucus. The change in composition may be due to an increased amount of undigested foodstuffs, which can sometimes be recognised macroscopically, but better still microscopically.

The presence of mucus is the most frequent and most characteristic symptom of intestinal catarrh. The amount of mucus voided varies greatly, however, according to the seat of the disease; this will again be referred to in the discussion on the localization of intestinal catarrh. The presence of mucus in one movement does not conclusively prove the existence of an intestinal catarrh, neither does its absence exclude the same. The first statement will scarcely be denied; it is important, however, to emphasize the fact that severe catarrhal conditions may exist without the production of mucus. Thus, since Nothnagel's[12] investigation on this subject, it has generally been accepted, and I believe with perfect justice, that in *atrophy of the intestine* (which, by the way, frequently eludes a positive diagnosis) mucus is entirely absent from the dejecta. An admixture of blood is not one of the characteristics of primary enteritis; it, as well as pus, represents only one of the complications.

The course of chronic enteritis is tedious and protracted. Undoubtedly a functional cure is sometimes achieved. If we consider analogous conditions of other organs (the stomach, the urethra, the larynx, the uterus), it is at least doubtful whether there are ever any cures in an anatomical sense. The following experience, which undoubtedly other physicians have also had, is very interesting, viz.: after a catarrh lasting for many years, a sudden change in its character may occur—i. e., chronic diarrhœa may gradually give place to a marked tendency toward constipation, or *vice versa*. In these cases there have been changes in the mechanism of innervation, the nature of which is as yet entirely unknown to us.

If we consider the picture outlined in the foregoing pages, the *diagnosis* of chronic enteritis in marked cases is not especially difficult. It is more difficult, however, in atypical cases, and particularly its differentiation from simple functional diarrhœas is an extremely delicate problem. There is no objective symptom in the latter that may not also be present in catarrh. From numerous personal observations, I can state that mucus, undigested food remnants, and bile pigment may be present in nervous diarrhœa as in intestinal catarrh. An analysis of such cases shows—and this is perhaps the key to the proper understanding of these complicated conditions—that, in consequence of a continued chemical and mechanical irritation of the intestinal mucous membrane, a true intestinal catarrh may be developed from a purely functional diarrhœa. For the elucidation of these difficult questions, an exact previous history and *status præsens*, particularly as to the nervous system, are very necessary. We thus learn that the disease occurs paroxysmally, that it is frequently increased by psychic excitement or mental and bodily fatigue, that during rest it subsides or intermits, and that also in the latter case an occasional dietetic error is borne without injurious results, etc. Objectively, other signs of hysteria or neurasthenia may be present: increased reflexes, local hyp- or hyperalgesia, autographia, vasomotor disturbances, etc.

In spite of all these diagnostic auxiliaries, we entirely agree with Nothnagel that the diagnosis can frequently be made only *ex juvantibus et nocentibus.* In discussing nervous diarrhœa, we shall again refer to the individual symptoms.

As indicated above, the abortive or ill-defined cases of intestinal catarrh are also of practical interest. These may occur with apparently normal intestinal evacuations, and manifest themselves by occasional painful abdominal sensations. A careful examination of the fæces will, however, reveal the characteristic signs of intestinal catarrh, now to be described.

For the diagnosis of intestinal catarrh, aside from the less positive data obtained from physical examination of the abdomen, there are in reality only two other methods of examination: test lavage of the intestine (see page 87) and the examination of the dejecta. The first method at once shows whether the large intestine is the seat of a chronic irritation attended with hypersecretion of mucus, and should for this reason never be omitted in any [suspected] case of intestinal catarrh. Under certain circumstances examination of the dejecta may decide the diagnosis; it may show the presence of abnormal undigested matter or of certain admixtures (especially mucus) as prominent constituents of the fæces. Regarding the undigested foodstuffs, we must at present be satisfied with the gross differentiation derived from the comparison of the normal with the abnormal stool. A positive functional test of the intestine alone will furnish us with an exact knowledge of the deviations from the normal.

For the examination of mucus and other abnormal constituents, see above, page 96 et seq.

DIAGNOSIS OF CATARRH OF THE SMALL INTESTINE

The main symptom of catarrh of the small intestine is the anomalous condition of the fæces. The subjective symptoms previously mentioned are also of some value.

With the exception of the dejecta (soon to be described), the most important, at times the most diagnostic objective symptom, is the presence of a well-defined pressure sensitiveness. In my experience, catarrh of the small intestine may sometimes, at first sight, be differentiated from that of the large intestine by this means. While objective tenderness is either entirely absent in the former, or, when present, is usually elicited only in the mesogastrium above or below the umbilicus (especially in tuberculous diarrhœa), diffuse tenderness on pressure is frequently found in chronic catarrh of the large bowel; although rare in the region of the cæcum, it is well-pronounced over the sigmoid flexure and descending colon.

In these cases the evacuations are usually diarrhœal. But, as Nothnagel has conclusively shown, they may be semisolid or even quite firm. Where they are fluid or semifluid, the admixture of mucous shreds, as well as the presence of bile pigment and of bile-tinged epithelial cells and muscular fibres, indicate an affection of the small intestine. Where the evacuations are firm, the question of disease of the small or of the large intestine is most readily and positively answered by means of a test lavage of the bowel. If the irrigation repeatedly brings away large quantities of membranous or viscid mucus, there is undoubtedly a catarrh of the large intestine present; whether catarrh of the small intestine coexists can not be determined by this procedure. If the test lavage results negatively, the diagnosis is very difficult in case the stool is firm or semisolid. Nothnagel has stated that the presence of so-called yellow mucous granules or of hyaline mucous islets is characteristic of catarrh of the small intestine, or of catarrh of the uppermost portion of the large intestine (page 96). The existence of these formations has, however, been denied by Ad. Schmidt, whose opinion I must corroborate on the ground of numerous personal examinations of fæces.

It will thus be seen that, aside from the subjective symptoms, which are for the most part uncharacteristic, the diagnosis of that form of catarrh of the small intestine which is accompanied by constipation or by normal dejecta, rests upon the demonstration of abnormal products—i. e., unchanged bile pigment (demonstrable either macro- or microscopically), the constant presence of exceedingly numerous muscular fibres, of well-developed starch cells, of fat in the form of globules or needles (fatty acids or fatty soaps) in the fæces.

As has been previously remarked, the localization of a catarrh of the small intestines (duodenum, jejunum, ileum) is very difficult, and usually impossible. Nevertheless, according to my experience, we occasionally meet with cases in which the diagnosis may be ventured. The following case is an illustration:

Emil B., sixty-two years of age, cabinetmaker, has always been perfectly healthy. He was taken ill in the summer of 1892 with a feeling of pressure in the epigastrium and violent diarrhœa (about seventeen evacuations in twenty-four hours). From October, 1892, to March, 1893, his condition improved. At this time there was a recurrence of his diarrhœa (four liquid stools a day), together with painful sensation of pressure in the region of the stomach. The patient complained also of shortness of breath and bodily weakness. His condition, as recorded at the time, was as follows : Examination of the abdomen, negative ; lungs emphysematous ; diffuse bronchitis ; heart dulness partially obscured by the lungs ; cardiac sounds clear. Test lavage of the intestine yielded numerous large shreds of mucus. Microscopic examination of fæces negative. Treatment dietetic. Improvement. On July 18, 1895, the patient returned, complaining again of diarrhœa. The first stool in the morning is the only one of a somewhat firm consistency; the others (six to ten) are always fluid. After the principal meal there are always two to three stools at short intervals. No pain or tenesmus is present ; rumbling of the bowels is felt. The appetite is fair; there are no eructations, no vomiting ; the diarrhœa is said to be unaffected by the kind of nourishment taken.

Condition on July 18, 1895. The patient is a well-nourished man, with a normal colour of the skin. The mucous membranes are somewhat pale. The lungs are emphysematous. The area of cardiac dulness is somewhat diminished ; heart sounds are clear, though somewhat muffled. The radial artery rigid. The abdomen is well rounded, the abdominal muscles tense. There is no tumour present, no tenderness, no splashing sounds, œdema, or eruptions of the skin. The urine is free from albumin and sugar, but contains indican in moderate amount.

The dejections are fluid, of a brownish-yellow colour ; their odour is pungently acid, but not at all fæcal. Reaction, *decidedly acid.* They give a positive reaction for bile pigment. A digestive test was made with a filtrate of the stool, by means of a scale of albumin (serum albumin), without any further addition. At the expiration of three and a half hours the albumin scale was perfectly dissolved.

Microscopical examination showed little mucus, much starch in well-preserved granules, many muscular fibres, strongly tinged with bile. Examination of further specimens of fæces gave the same result, excepting that the digestive test (with albumin) was negative. Test lavage of the large intestine yielded jellylike mucus.

My opinion that this case was one of catarrh of the upper portion of the small intestine (with probable catarrh of the colon) is based upon the pronounced biliary character of the brownish-yellow fluid evacuations, which were quite free from fæcal admixture. Although the digestion test only gave positive results on a single occasion—something very unusual in my experience—yet this, when taken in connection with the other features of the case, serves as an additional support to the diagnosis. As far as I have seen, Nothnagel[12] is the only one who has called attention to the peculiarities of the stools in such cases, and to the difficulties of a differential diagnosis between these diarrhœas (jejunal diarrhœa) and the true catarrhs.

Special mention should be made of a form of dyspeptic diarrhœa which I have frequently met with, and which is of extreme practical importance. It is associated with severe types of chronic gastric catarrh, such as, following the nomenclature of Einhorn, are now called achylia gastrica. Biedert[13] published minute and valuable personal observations, and subsequently Einhorn[14], and especially Oppler[15], have called attention to this condition. It is characterized by a subsidence of the subjective gastric symptoms and the appearance of chronic intestinal disorders, severe diarrhœa being especially complained of. As a rule, while the motor function remains intact, the gastric secretion completely disappears. Examination of the stools shows a disturbance in digestion, abundant unaltered food residues, remains of animal tissues, especially connective tissue, vegetable products, and also, but less often, fatty elements are present. Of the numerous observations of this sort which I have collected in the course of years I will quote but one, and that notable for the extent of the functional disturbances.

Max G., nurse, forty-one years old. Underwent an operation for hemorrhoids six years ago, after which—doubtless because of a stricture of the rectum—rectal bougies were passed for several weeks. From that time until two years ago the patient remained well; then he began to have occasional attacks of diarrhœa. The patient denies any, even slight, gastric symptoms at this period, excepting perhaps acid eructations. At all events, he could eat any kind of food without discomfort. In the last two months the diarrhœa has markedly increased, so that he now has four or five thin fluid movements each day, without tenesmus.

Examination of the abdomen and rectum is quite negative. On the other hand, repeated examination of the gastric contents shows absolutely undigested food with a good deal of mucus, and neither free nor combined hydrochloric acid. Examination for pepsin gives negative results; that for rennet ferment shows that only when undiluted is the gastric juice capable of coagulating milk. In order to ascertain the digestive capacity of the intestine, the patient was given a test meal on the evening of December 19, 1898, consisting of a quarter of a pound of uncooked beef cut into small pieces. On the following morning the fæces consisted *almost exclusively of undigested meat, particularly the connective tissue.*

At a second test, in which the same quantity of beef was given in minced form, numerous undigested fragments of connective tissue were again found, and on microscopic examination a moderate quantity of muscular fibres. This stool was a thin fluid of brownish colour; its filtrate gave a flocculent precipitate with nitric acid, and with caustic potash and sulphate of copper (in very dilute solution) a decided biuret reaction. The examination was repeated on several occasions with the same result, except that the biuret reaction varied in intensity.

This case is therefore one of those which the older writers used to call lientery (λεῖον, slippery, and ἔντερον, bowels). It would be difficult to decide in this case whether we have to deal with a true enteritis or with a partial atrophy, but doubtless a marked functional disturbance in the secretion of the intestinal glands and the pancreas must be thought of. The latter feature has acquired special interest from the fact, long since known, but recently confirmed by Pawlow[16] (who has demonstrated it in a way worthy of imitation by other investigators), that the hydrochloric acid of the gastric juice is the most important excitor of pancreatic secretion. Thus, if we may transfer the results of animal experiments to human physiology, a new light is shed upon the origin of this form of intestinal dyspepsia.

Diagnosis of Chronic Catarrh of the Large Intestine

In well-marked cases the diagnosis of uncomplicated catarrh of the large intestine seldom offers difficulties. The subjective signs are essentially those which depend on the function of defecation, and are not characteristic, unless it may be the tormenting feeling of pressure or pain, above mentioned, or, in diarrhœal cases, the colicky pains, to which tenesmus may be added, if the process extends to the lower segments of the bowel.

The objective signs consist in tenderness on pressure and changes in the fæces. The tenderness on pressure, which has already been described, is by no means constant. When present, it seldom extends over the entire length of the large intestine, but affects localized segments, most frequently the descending colon and sigmoid flexure. The sensitiveness may be of various degrees of intensity; occasionally even the slightest pressure is disagreeable. Patients with well-marked tenderness usually complain of pain in the corresponding portion of the bowel, particularly after being seated for a long time, or after walking, bending, or riding, etc.

The nature and form of the mucus evacuations is of great importance in determining the regional extension of catarrh of the large intestine. The lower the portion affected, the purer and more unmixed are the mucus dejections. Thus the passage of pure mucus points to catarrh of the rectum, the sigmoid flexure, or the lower segment of the descending colon. Similarly, hard masses of fæces coated with mucus indicate that the disease is situated in the lower portions of the bowel.

The examination of the stools gives the following results:

(*a*) *With Constipation.*—The stools frequently show mucus either gelatinous or in strips (membranes), but not so constantly as after irrigation. In other respects the formation may be normal or diarrhœal, or of the consistence of pulp, or like sheep dung. For the microscopical peculiarities, see chapter on the Examination of the Fæces.

(*b*) *With Diarrhœa.*—In these cases the diagnosis as to localization is best reached by exclusion—that is to say, by observing the absence of unchanged biliary pigment. In other respects the stools in this, as well as in the previously described form, show only scanty food residues, in particular very little unchanged muscular fibre, starch, or fat. The mucus is usually found intimately mingled with the stool, or in the form of little shreds visible when the vessel is rotated.

Diagnosis of Mixed Forms of Intestinal Catarrh

The diagnosis can be made solely from the peculiarity of the stools, which in such cases are almost always diarrhœal. As before, the recognition of biliary pigment, undigested food residues, especially muscular fibres, points to a localization in the small intestine, while the results of irrigation will show whether or not the large intestine is involved. The character of the stools as regards mucus resembles that of catarrh of the large intestine.

Although in the preceding pages we have described the clinical characteristics of intestinal catarrh with especial reference to the determination of the regional extent as fully as our present knowledge permits, we must not forget that such a delineation is largely schematic. When we consider that hypertrophic catarrhal processes are frequently associated with atrophic changes that can not be diagnosticated, that catarrhs may arise from neurotic conditions, that disturbances in intestinal absorption must be considered and may markedly influence the clinical picture, we must exercise the greatest caution in expressing an opinion in those cases where the existing conditions permit of more than one interpretation.

Treatment of Chronic Enteritis

In the treatment of primary chronic intestinal catarrh, as in all catarrhs of mucous membranes, the same principle that we have insisted upon in acute enteritis holds good: *Protection of the diseased organ.*

Especially must the nature, the seat, and the extent of the underlying disease be considered in this connection. In the absence of better criteria the character of the stools will serve as an indicator, in all essential features, of the morbid processes going on within the intestine.

For all practical purposes we are called upon to treat but three conditions: Diarrhœa, constipation, and constipation alternating with diarrhœa.

1. *Diarrhœa*

The most important part of the treatment is to insure complete bodily rest and protection. In the milder cases, moderate physical exertion or intellectual work may be permitted, but in the severer forms these should be absolutely prohibited. *Such patients should be put to bed and kept there for at least several weeks.*

Absolute rest in bed has the advantage that, once for all, it puts a stop to the external influences so often conspicuous in chronic diarrhœa, namely, cold, wet, mental or physical exertion, and, above all, dietetic excesses or transgressions. As the other diagnostic and therapeutic procedures are difficult to carry out at home, the treatment—especially of advanced cases—is best conducted in hospitals or in private sanitaria, in which dietetic arrangements are well managed.

During the stay in bed, continuous warm fomentations may be used with advantage. An appropriate *diet* is absolutely essential. There are three points to be observed: 1. To avoid injurious substances which might increase the intestinal catarrh. 2. To select such nutrients as will be assimilated in the particular case. 3. To combine the greatest variety of those nutrients whose physiological action is astringent or which reduce secretion—in other words, those which have a tendency to constipate.

The details of such a diet have been given *in extenso* in the General Section (page 149). For those physicians who have had little experience in prescribing a special dietary, the following may be of assistance:

8 A. M.—"Eichel cocoa" (in water), one saccharin tablet (or crystallose), toast and butter (20 to 30 grams).

10 A. M.—One cup (200 grams) rice gruel, buckwheat or oat grits in veal bouillon (avoid salt). In addition, 50 grams roasted veal or beef (scraped), or fried fish or cold meat (avoid salt or strongly pickled ham).

1 P. M.—Soup of peas or beans or *purée* of oatmeal, farina, or corn starch, etc. (addition of nutrose, tropon, or eucasin allowed; somatose forbidden). In summer, huckleberry soup (with saccharin, if desired). Two hundred grams of rice bouillon (avoid rice with milk),* or farina bouillon, well thickened by cooking.

Green vegetables or potatoes in *purée* form (50 to 100 grams).

Meat and fish (fat excepted), 50 to 100 grams. (Butter sauce allowed; cream sauces or highly seasoned sauces forbidden.)

Stewed fruits, with the exception of huckleberries and cranberries, forbidden.

Custards (corn starch, with a little yolk of egg and saccharin) allowed. (Avoid fruit juices.)

As beverages: Huckleberry wine, Burgundy, Camarite, Simaruba wine, old Bordeaux. (Sweet wines, white wines, and effervescent beverages forbidden.)

4 P. M.—Tea (without milk), with saccharin or cocoa. Cakes, toast, zwieback (with butter).

7 P. M.—Strained gruel (oatmeal, etc.). Cold or warm meat (50 grams), toast, butter (20 grams).

One or two glasses of huckleberry wine.

9 P. M.—One glass of huckleberry lemonade,† warm, or hot mulled wine (saccharin), or tea, with red wine.

It may be necessary to cut out many articles from this dietary. It should only be relaxed when the general and local conditions are satisfactory, and the stools have been formed for at least four weeks without any relapses having occurred.

Sugar, pastry, milk, organic acids, salt, beer, effervescing beverages, cold drinks, and fruit ices should be positively avoided for months, and in many cases for years.

The value of such a diet, taken in connection with absolute rest in bed, can not be overestimated. Indeed, I know of nothing which can replace this treatment. Nevertheless, in some cases the use of medicines will be required.

Medicinal Treatment.—We know of no drug which will cure

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* As to avoiding milk, see page 144.

† Huckleberry lemonade is made by adding one or two teaspoonfuls of huckleberry jelly to boiled water.

an intestinal catarrh; but one of the symptoms, diarrhœa, may sometimes be favourably influenced by appropriate medication.

There is nothing to add to what has been said in the General Section (page 149 et seq.). We can only repeat that we have little faith in the value of the numerous astringent and antiseptic remedies which have recently come to notice. Yet there is one drug that should not be forgotten, because it combines a certain degree of utility with the absence of any objectionable feature. I refer to chalk. It should be given as a mixture of equal parts of carbonate of lime and phosphate of lime. For several years I have preferred to treat cases in which diarrhœa has persisted in spite of dietary regulation, by giving a teaspoonful of this powder three times a day.

Jaworski[17] has recently recommended chalk dissolved in carbonated water for diarrhœal cases. He uses two formulæ, a stronger and a weaker, as follows:

℞ Calcii carbon 2.0
Calcii salicyl............................. 2.0

Dissolved in one litre of highly charged carbonic water. (Aq. calcii mitior.)

℞ Calcii carbon 4.0
Calcii salicyl............................. 3.0

In similar solution. (Aq. calcii fortior.)

One half a glass of the stronger is to be taken fasting, in the morning, and a half glass of the weaker, three times a day, after meals. In severe cases Jaworski recommends that the above be taken mixed with a half glass of warm Carlsbad Sprudel water.

The use of preparations of chalk is specially valuable when there are eructations of hydrochloric acid, for here the sodium preparations are contra-indicated on account of their laxative tendency. A combination of the above-mentioned chalk mixture with bismuth has been highly recommended. I prefer the beta-naphtholate (orphol). The following formula is unobjectionable and appropriate:

℞ Calcii carbon.
Calcii phosph., āā....................... 25.0
Bismuthi beta-naphthol................... 5.0

M. One teaspoonful three times a day.

Besides the preparations of lime just mentioned, the *natural mineral waters containing lime* are useful as adjuvants [see pp. 163 and 164]. They are especially suitable as table beverages. I have often ordered them (warm, one glass morning and evening) with good results, and can heartily recommend this treatment.*

Concerning the other hydrotherapeutic methods, see General Section, page 158 et seq.

For the functional diarrhœas that occur with achylia gastrica, Oppler[15] has found hydrochloric acid in large doses (20 to 30 drops, and even more) very satisfactory.

If there are gastric disorders, and especially loss of appetite with chronic intestinal catarrh, I particularly recommend the use of wine of calumbo (a dessert glassful three times a day, before meals), or the fluid extract of calumbo (a teaspoonful three times a day, in a wineglass of lukewarm water or in a wineglass of the above-mentioned solution of chalk).

The treatment of chronic diarrhœas by enemata is suitable in catarrhs of the colon and the rectum. In addition to those already described (page 181), I have seen excellent results from enemata of bismuth (a teaspoonful in 250 cubic centimetres of water). This is analogous to Fleiner's method for the treatment of gastric ulcer.

2. *Constipation*

In the treatment of constipation, bodily and mental rest may contribute toward a good result; only in very severe cases is absolute rest in bed indicated. Experience shows that warm, frequently repeated fomentations have a favourable effect upon the pain.

The chief indication to be met in catarrh of the intestine accompanied by constipation is the removal of this symptom by appropriate diet. In all essential features it is the same diet as that to be fully described in the chapter on Constipation, but with this important exception, that *all foods which are rich in cellulose, or other indigestible substances, must be avoided.* If, as is usually the case, we succeed in regulating the evacuations by the diet, the abnormal secretion of mucus gradually disappears without further treatment (see chapter on Membranous Enteritis). In these cases I do not believe purgatives should be given; indeed, I have a suspicion that their use may cause, or at least aggravate, an intestinal catarrh. If directions as to diet do not suffice, I recommend mild enemata of rape-seed oil, oil of sesame, or olive oil; of neutral soap (5 grams to 250 cubic centimetres of water), castor oil, cod-liver oil, soda, etc. (see page 179). Irrigation for the purpose of cleansing the intestine of mucus is a very useful adjuvant. The following solutions are to be recommended for this purpose: Lime water (3 to 4 tablespoonfuls to 1 litre of water), carbonate of soda (1 dessertspoonful to 1 litre of water), Carlsbad salt (in the same proportion). Other desirable agents for the same purpose have been mentioned on page 181.

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* It is much to be regretted that the treatment of chronic intestinal catarrhs has not been undertaken at the above-mentioned springs. With appropriate installations for providing suitable diet (somewhat after the manner of Carlsbad), these springs would, in my opinion, take a prominent place in the treatment of intestinal catarrh.

3. *Constipation, alternating with Diarrhœa*

The same principles hold good as in the form just described. It is only necessary to decide which is the primary or predominating feature, and this can readily be ascertained by the use of a test diet for a few days.

3. Membranous Enteritis

Preliminary Considerations.—By membranous enteritis, or, still better, membranous colitis, we understand a form of catarrh of the large intestine which is characterized by three cardinal symptoms: (1) A peculiar mucous formation; (2) anomalies of intestinal function; (3) painful spasm of the intestine. In addition, there are a few other collateral symptoms, which, however, have nothing to do with the clinical picture proper of the disease.

The classification of membranous colitis with chronic enteritis does not altogether correspond to the views which prevail at the present time, especially among German authors. The predominant conception is that which Siredey[18] seems to have been the first to advance in 1869—that the membranous mucous formation was the result of a peculiar secretory neurosis. Da Costa[19], to whom we owe its first classical description, looked upon membranous colitis as of nervous origin. The German authors, in so far as they have expressed themselves concerning the pathology of the disease, do not agree. von Leube[20] and Rosenheim[21] are inclined to regard it as a neurosis of secretion. Ewald[22] takes a middle ground, and Nothnagel[23] makes a sharp distinction between mucous colitis and mucous colic, one having an anatomical and the other a functional basis. The standard French authors (G. Sée[24], Potain[25], Alb. Mathieu[26], de Langenhagen[27]) rather incline to the view that there is a superficial catarrh. On the other hand, American authors (Mendelson[28], Einhorn[29], and others) have recently laid stress on the nervous character of the affection. Vanni[30] also speaks of a myoangioneurosis of the intestine with hypersecretion of mucus. As far as I have seen, the latter idea has made a deep impression in medical circles, and has had an undeniable influence upon treatment.

From personal observation I can say that it is difficult to come to a decision. There are cases in which the nervous, restless character of the affection is very prominent, so much so that it is difficult to believe that it has a material basis. On the other hand, there is no doubt in my mind that membranous colitis is frequently found in patients who are not at all neurotic, or in whom the neurotic stigmata are positively or probably the results of the disease. An unprejudiced estimate of the frequency of the two groups leads me to the opinion that the latter predominates.

This much is certain: the idea that membranous enteritis is one of the phases of hysteria or neurasthenia must be rejected as too sweeping.

Unfortunately, neither experimental pathology (Vanni[30], Akerlund[31]) nor pathological anatomy affords us much assistance as to etiology. We have but two clinical observations followed by autopsy—those of O. Rothmann[32] and of M. Rothmann[33]. In the former, although the entire intestinal tract was carefully examined by C. Ruge, nothing abnormal was found; while in the second case, reported by the younger Rothmann,* all the characteristic lesions of a catarrh of the large intestine were demonstrated by a very thorough histological examination. Although it may seem venturesome to draw any conclusion from two findings so diametrically opposed to each other, yet there is no doubt that the positive result has much greater significance than the negative one.

Aside from the main question whether we are dealing with a functional or an inflammatory condition, there are a number of other etiological factors to be considered. First of all, the influence of habitual constipation must be emphasized. According to the experience of most authors, habitual constipation is one of the most constant affections occurring together with membranous enteritis. A few (Ewald[34], Einhorn[29], and others) call attention to an antecedent diarrhœa as a cause. I have also seen such cases, but only after the use of astringent enemata. In addition, numerous observers (Glénard[35], A. Mathieu[26], Ewald[34], Boas[36], Akerlund[31], de Langenhagen[27], Einhorn[29], and others) have accentuated the relationship between coloptosis and membranous enteritis. This etiological factor has a certain influence, but only in so far as it favours the establishment of habitual constipation.

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* The report of O. Rothmann does not state whether a histological examination was made.

Attention has been called by French investigators to the relation between membranous enteritis and uterine diseases (Ozenne[37], Letcheff[38], and others); but these observations seem rather to relate to accidental complications. It is well known, moreover, that uterine diseases, as well as abnormal conditions of the adnexæ, may cause constipation by compression or adhesions, and thus predispose to membranous enteritis.

Finally, at a recent date, various French investigators (A. Mathieu[39], de Langenhagen[27], Chevalier[40]) have laid stress on a certain connection between membranous enteritis and intestinal lithiasis. The periodical formations of gravel, accompanied by severe colic, which had already been recognised and which was recently redescribed by Dieulafoy[41], are supposed by Mathieu[39] to be a constant feature of membranous enteritis. All that has been published on this subject simply goes to show a possible coexistence of intestinal lithiasis with the disease under consideration, but no proof has yet been offered that there is any etiological relation.

Artificial membranous enteritis is a very important condition, which from a practical standpoint has received much less attention than it merits. I have frequently observed it after enemata of tannin, alum, glycerin, and nitrate of silver. In some cases the clinical picture of membranous enteritis already existed (see Case IV); but I have become convinced that the symptoms may be kept up and increased by irritant injections. Membranous enteritis is also observed as a sequel to acute enteritis. It is doubtful whether this condition is altogether identical with the one now under consideration.

After these preliminary remarks, we will proceed to

Symptomatology and Diagnosis

When we analyze closely the description of the symptoms of membranous enteritis, as given by the most prominent authorities (Da Costa[19], von Leyden[42], Nothnagel[12], Kitagawa[43], Krysinski[44], A. Mathieu[39], Germain Sée[24], de Langenhagen[27], and others), we find such a lack of agreement that the question arises whether these authors are dealing with the same affection. In some of the cases it can be shown with certainty that the clinical picture belongs essentially to the group of colica mucosa. Others are complicated by gastric atony, intestinal prolapse, appendicitis, etc. In still others uterine complications exist. In one case, recently described by Henschen[45], larvæ of the fly were present in the intestinal canal. Others were undoubtedly due to artificial influences such as have been just mentioned. Finally, there are several cases (Marchand[46], O. Rothmann[32], Richardière[47]) which, as far as concerns the bowels, gave no symptoms during life.

Nor does this exhaust the list; it would take too long to allude to all of them. We can only say that for diagnostic purposes membranous colitis is sometimes an independent disease, and sometimes is found in conjunction with other affections. It is thus easy to understand that the clinical picture presents manifold exceptions and variations.

I have thought it advisable, therefore, to select the following from the large number of cases which I have recorded, and in connection with them to discuss the diagnosis:

CASE I. *A case of membranous enteritis, with severe disturbances of nutrition, which had existed for many years. Permanent cure.*

Mrs. Regina B., twenty-seven years old, born in Poland, and for several years a resident of Berlin.

The patient states that for seven years she has suffered from loss of appetite, eructations, pains in the stomach after the ingestion of food, flatulence, and persistent constipation. For some time past enemata have nearly always been required to obtain movements from the bowels, and the patient has often noticed membranous and tubular masses of mucus in the stools. These have sometimes appeared in such abundance that the stools consisted of almost nothing but them. The patient asserts that during this period she was very nervous and uneasy. *She does not, however, remember having had any marked pain immediately before such mucous stools.* At her first visit to the polyclinic (March, 1893) she looked very ill, was extremely emaciated, so that incipient phthisis was suspected, but not confirmed by examination. She was treated by irrigations of the intestine; the washings frequently showed masses of mucus, membranes of mucin, and tubular casts of various calibres. The subsequent treatment was wholly dietetic ("constipation diet").

Under the latter treatment and a course of waters the patient's condition improved materially; she gained in weight to a considerable extent, and began to get a healthy colour.

At my request she presented herself in March, 1895, and again in February, 1899, for examination; she stated that her health had continued good, and that there was no longer constipation. Irrigation showed that the mucous masses were no longer present.

CASE II. *Severe form of membranous enteritis, complicated by disorders of the stomach and bladder. Cure.*

Mrs. B., widow, Kloster Lehnin, near Brandenburg, thirty-six years old. The patient states that since the death of her husband and a sixteen-year-old daughter she has been very nervous and irritable, sleeps badly, complains of tremor and spots before the eyes.

The patient has suffered from extreme constipation since childhood; has always used enemata and laxatives. In the last three years disorders of the stomach have appeared: poor appetite, nausea, but no vomiting, discomfort, and pressure in the epigastrium. Gradually she began to have attacks of severe pain in the epigastrium after eating, which came on even after a spoonful of milk. This pain radiated toward the sides and back, and was more severe after food not easily digested. The constipation increased so that four tablespoonfuls of castor oil and an enema of oil produced only a small evacuation. There was marked emaciation, and the patient was confined to bed for months. In the winter of 1895–96 she first noticed that after enemata pure mucus was passed, sometimes in the shape of little shreds, and sometimes larger aggregations in tubes or bands as long as half a metre. After passing these the patient used to feel better. With these complaints *there was associated pain in the bladder from time to time, ending in the passage of a light-coloured urine of low density.* On admission to the clinic in May, 1897, she complained of debility, weakness on walking, pains along the spine, anorexia, slight eructations, severe constipation, and the passage of mucus in the stools.

Condition on admission, May 5, 1897 (with the omission of unimportant features): Floating kidney on the right side; fundus of the stomach at the level of the umbilicus. Loud splashing sounds in both iliac fossæ, and to some extent also in the epigastrium.

An evacuation followed irrigation of the intestine; it resembled sheep dung, and was covered with small shreds of mucus; it was small in quantity, and of a brown, or perhaps greenish brown colour.

Palpation of the abdomen showed that the left iliac region was very tender; there was also slight tenderness in the epigastric region.

A second irrigation on the same day showed abundant masses of mucus, several of the shreds measuring a few centimetres in length. They were white or yellowish brown in colour, some membranous, and some vitreous in appearance.

Repeated irrigations gave the same result. The microscopical examination gave the usual findings. The stools were in other respects normal. Treatment: constipation diet and intestinal irrigations.

In spite of these measures the constipation was not entirely relieved. Enemata could not be dispensed with. In the further history of the case the pains in the stomach and spine disappeared and the patient gained in weight. The abdominal pains were felt occasionally. The evacuations obtained by enemata frequently contained large shreds of mucus. The neighbourhood of the sigmoid flexure was still sensitive to pressure, but much less so than formerly. Treatment, aside from diet, consisted in pulv. glycyrrhizæ comp., one teaspoonful twice daily.

June 24, 1898.—The patient now has regular movements from the bowels; the pains have almost entirely vanished; weight and strength have increased. The evacuations are free from mucus. The region of the sigmoid flexure only slightly sensitive.

CASE III. *Membranous enteritis, with severe constipation and occasional acute colics. Previous history of ulcer of the stomach (or duodenum?).*

Mrs. B., resident of G., forty-one years old. In her nineteenth year, four weeks after marriage, the patient suffered from acute peritonitis (gonorrhœal infection ?). She was confined to bed for seven weeks, and had pains in the abdomen for some time afterward.

As a girl she suffered from constipation, which became worse after the attack of pelvic peritonitis. Two years later she had an attack of pleurisy on the right side, following which she began to have a persistent gastric pain. In the year 1885, after the appearance of tarry blood in the stools, a gastric ulcer was diagnosed. In 1887 there was another attack of melæna, and in 1890 vomiting of blood. She underwent von Leube's treatment for ulcer, at Würzburg. Improvement resulted, but only under the strictest diet. Mucus was first noticed in the stools in 1888, the bowels being extremely constipated. The same fact was noted during the treatment for ulcer in 1890. Gradually severe pains developed on both sides of the abdomen at the level of the umbilicus; these were only relieved by a free movement of the bowels. Sometimes nothing but mucus was passed, and at other times it was accompanied by fæces. When mucus only was passed there was no alleviation of the symptoms. Such mucous evacuations occurred every four to six weeks. In the intervals mucus was either absent or only found in small quantity.

Present condition: Sensitiveness on pressure localized at 1½ centimetres to the right of the median line at the junction of the middle and lower thirds of the space between the xiphoid cartilage and the umbilicus. Intestinal area quite free from sensitiveness. Irrigation of the intestine on two occasions gave only a very small quantity of mucus.

CASE IV. *Development of membranous colitis during pregnancy, with severe pyrexia. Normal labour. Cessation of the fever. Continuance of the colitis. Cure by producing regular movements of the bowels.*

Mrs. H., twenty-five years old. The patient, who was healthy, with the exception of a varying degree of constipation, suddenly took sick in the sixth month of pregnancy, on June 19, 1898, with a chill and a fever reaching 39.6° C. [103.3° F.]. She complained of pain in the lower part of the abdomen on the right side, which pain radiated to the lumbar region and down the right leg. Examination yielded no positive results. The illness during the following fourteen days presented the picture of a septic infection.

In the beginning of July the fever and chills abated, followed on the 10th by symptoms similar to those at the onset. About the middle of the month the stools for the first time showed mucous casts, which were both tubular and ribbonlike, and 10-12 centimetres in length.

For the following particulars I am indebted to her family physician, Dr. Laux, of Oldenburg:

On the 18th of July, having just returned after an absence from town, I saw these evacuations for the first time. Though quite fresh, they had a penetrating odour, as if in a state of decomposition, which was also suggested by their dirty, grayish green colour. By means of two or three large enemata, I succeeded in removing more or less abundant masses of fæces, with a resulting subsidence of the strong odour, and a return to the usual colour. Under this treatment the chills and fever disappeared, and the subjective symptoms improved to some degree. On August 1st delivery occurred, setting in with a chill and fever, but otherwise normal in its progress. The puerperal period was uneventful. Since then there have been, off and on, evacuations of mucous shreds, but finely broken up and in small quantity. On the day before such passages there is pain, which is relieved by the evacuation. Persistent constipation exists, but is controlled by enemata. Regulation of the bowels by constipation diet in my clinic resulted in a complete arrest of the mucous discharge.

The reported cases of membranous enteritis show an extraordinary preponderance of the female over the male sex. All the authors agree on this point. Litten found 80 per cent and Kitagawa 90 per cent in women; Einhorn found, in a total of 20 cases, 2 men and 18 women, or about the same relation as the last-named author. This is not surprising when we consider the prevalence of coloptosis and constipation among women.

The greatest number of cases of membranous enteritis occur during the second, third, and fourth decades of life; it is only rarely found during the later years of life, and rarest of all in childhood. A few cases have been reported in the newborn (Longuet[48], Ullmann[49]) and in the early years of childhood (Löwenstein[50]). I have seen a well-marked case in a two-year-old girl of very nervous temperament.

The chief complaints of the patients relate to disturbances of the intestinal functions. Constipation is, as I have repeatedly said, the condition in the majority of cases; diarrhœa is certainly atypical. Paroxysmal pains are a very frequent symptom. They usually mark the onset of attacks, and are of an exceedingly acute colicky type, so that they often cause symptoms of collapse. The attack ends with the passage of fæces and membranous mucus, or of the latter alone. The stools have the usual appearance of spastic dejections. These intestinal colics are, however, by no means a necessary symptom; they may be slight, or entirely absent, or there may be colicky pains without passage of mucus; or, finally, membranes may be passed with or without attacks of pain. If we are successful in regulating the bowels, the painful intestinal spasm and the mucous evacuations usually cease, or appear only occasionally and to a limited extent.

Together with these symptoms there may be various other complaints, partly of a nervous and partly of an organic origin; these have nothing to do with the disease as such; they are nothing but accessory symptoms or complications.

In typical cases the objective signs are sensitiveness over the colon or portions of it, and the passage of mucus.

In the majority of cases the sensitiveness is noted over the descending colon or the sigmoid flexure; in other cases over the cæcum and ascending colon, and apparently very much less frequently over the transverse colon. This sign alone is not characteristic of membranous colitis, since, as we have noted above, it is met with in ordinary catarrh of the large intestine; but in the former affection it is much more pronounced than in simple catarrh. The tenderness which under certain circumstances is not much less than in appendicitis, seems to bear a positive relation to the process, for it disappears or diminishes as cure or improvement occurs, to return in a surprising way when a relapse occurs.

The expulsion of mucus or membranes is the most decisive clinical sign. It is hardly conceivable that these formations should be mistaken for tapeworm, food residues, etc., or anything else, or that the affection should be confounded with croupous enteritis, if careful macroscopic and microscopic examination is made. Three kinds of mucous formation can be distinguished: 1, unformed, structureless mucus; 2, hyaline, tubular formations, which under certain conditions form a cast of the internal surface of the intestine; 3, membranous mucus, sometimes firm and sometimes spongy in consistence. The chief constituent of these secretions—for there is no doubt on this point at the present day—is mucus and an albuminoid body, the latter depending upon the varying admixture of cellular elements. According to careful investigations of Kitagawa[43], M. Rothmann[33], Akerlund[31], Ad. Schmidt[51], and Pariser[52], there is no fibrin present, and they are thus differentiated from the exudates in intestinal diphtheria.

In doubtful cases a microscopic examination is a useful supplement to the results of the macroscopic investigation. Even without the addition of acetic acid, but better with it, there will be found the peculiar threadlike substance in which cells, nuclei, and detritus are embedded in variable quantity. Most of the cells have lost their characteristic appearance (see Fig. 16, page 118), fresh, unchanged cells being rarely found. Opinions differ as to the cause of this degeneration. Nothnagel[12] thinks it is due to desiccation. Kitagawa regards it as a degeneration process (coagulation necrosis). As Ad. Schmidt[51] has recently shown, it is due to an infiltration of fatty soaps, on the removal of which the cells regain their bright, transparent appearance. There are also found a greater or less number of leucocytes, occasionally Charcot-Leyden crystals, and micro-organisms of various kinds. None of these have any special significance.

Complications are very frequent. Associated disease of the uterus and adnexæ and intestinal lithiasis have already been mentioned. The literature of the subject shows that albuminuria, pyrexia, epileptic attacks, tachycardia, dyspnœa, neuralgia, tremor, somnolence, amblyopia, and melancholia are occasional accompaniments. Einhorn[29] found that out of twelve cases achylia gastrica was present in five, and in several cases the gastric motor function was increased.

The *course* of membranous colitis, like that of habitual constipation, is exceedingly chronic, but like it, shows marked remissions and intermissions. The general nervous symptoms follow very closely the increase or diminution in the colicky attacks.

The *diagnosis* of membranous colitis can be made in most cases from the symptoms which have been detailed, especially from the results of repeated intestinal irrigation. This should not be postponed in any case. It will show—and I particularly insist upon this—that the membranous formation does not appear occasionally or suddenly, but that smaller or larger masses of mucous or tubular formations may often be identified during the intervals between the attacks. Some difficulty exists in the differentiation between simple colitis and the membranous variety, for we meet with cases which might with equal propriety be put in either class. In general, those cases in which actual membranes are passed should be put in the present class, and the remainder classed with the other forms of enteritis. The other fact already mentioned, that membranous enteritis may arise from artificial causes, must again be emphasized and should always be borne in mind.

TREATMENT

Until recently very unfavourable or doubtful results were obtained from the treatment of membranous colitis, and at the present time the disease is frequently obstinate, and yields reluctantly to therapeutic influences. This is especially true of those cases in which a hysterical element is prominent.

In cases of a conspicuously catarrhal type much more may be expected from treatment.

In accordance with our conception of the nature of the disease, chief stress is to be laid upon the treatment of the enteritis, without underestimating the importance of such elements as neurasthenia, enteroptosis, anæmia, or faulty nutrition.

We have already said, and it has been recently emphasized by von Noorden[53], that the chief feature of the treatment of membranous enteritis consists in the relief of the constipation by an appropriate diet. von Noorden[53] rather recklessly states that one rich in coarse constituents, with abundance of butter and fats, is appropriate. I have not had any experience with this method, but I can not suppress the thought that such a coarse diet may gradually set up intestinal irritation. I still believe in the view, expressed some time ago[54], and repeated recently, that a constipation diet, without husks or cereals, is the only suitable and successful dietary in membranous enteritis. Einhorn[29] has very recently taken a similar position.

It is not necessary to enter here upon the details of this diet, as it differs very little from that to be described in the chapter on Constipation. We agree with von Noorden as to the cardinal importance of an abundant supply of nutriment, since the patients are generally individuals who are depreciated by anæmia, faulty nutrition, or frequent pregnancies. I have seen excellent results from the employment of forced nutrition, always, of course, with due attention to the question of constipation.

In those cases in which the constipation is overcome by diet, local treatment of the intestinal tract is superfluous; in other cases it may be of advantage. According to Fleiner, enemata of oil are very successful, but I have not had sufficient experience with them to be able to say whether their effect is lasting. Careful irrigation with unirritating substances, such as physiological salt solution, sodium carbonate, or Carlsbad salt, may be of material assistance. On the other hand, caution must be recommended in the use of astringent solutions which are apt rather to increase the difficulty (tannin, alum, nitrate of silver, etc.). For the same reason I consider the stronger purgatives contra-indicated, while the milder laxatives, such as rhubarb, tamarinds, liquorice powder, and preparations of sagrada, may be of use if dietetic measures are not sufficient. Other drugs (ext. fl. hydrast. canadensis, bromids, opiates) have been recommended, but a real influence upon the morbid process is hardly to be expected from them. Perhaps the painful colics may be ameliorated by suppositories containing codeia, belladonna, or opium. In general, the continuous or intermittent application of moist, warm, or hot poultices, in connection with aromatic infusions, will suffice.

In cases of membranous colitis with prominent nervous disturbances hydrotherapeutic procedures are very valuable (half baths, shower baths, douches, wet packs, etc.). Change of air and a warm climate sometimes contribute to a cure. The mineral waters do not promise great or permanent results.

Among the curiosities of treatment, I may mention that the surgeons have attempted to cure membranous colitis by the establishment of an artificial anus (Hale White and Golding Bird[55], F. Franke[56]). They claim to have had successful cases.

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CHAPTER XIV

HABITUAL CONSTIPATION. DISPLACEMENTS OF THE INTESTINES[*]

A. HABITUAL CONSTIPATION

Preliminary Remarks.—By habitual constipation we understand a condition in which the intestine irregularly or incompletely evacuates its contents. An estimate of the normal frequency of defecation is intentionally omitted from this definition, for this may be greater or less without giving rise to a morbid condition. Every physician of experience knows, and every text-book of special pathology mentions, examples of extraordinary infrequency of defecation without disturbance of health. Less known and appreciated, and doubtless not so common, is the physiological occurrence of the opposite condition—that is, an unusual frequency of defecation, the stools being otherwise normal. Of this I have observed several examples.

There is a sharp line to be drawn between this condition and morbidly retarded defecation with its train of consequences. The latter is sometimes an acute and sometimes a chronic condition; it may be artificial, or it may result from alimentary causes; occasionally it accompanies or is the sequel of other not wholly intestinal disorders; finally, it may be idiopathic, or, to speak more accurately, be independent of any recognisable organic cause.

Of the forms just mentioned, alimentary constipation, on account of its great practical importance, deserves a few remarks. It owes its origin, no doubt, to a perverted or insufficient diet. It is specially observed among the higher classes where there exists a very obstinate traditional preference for what is called a "nourishing diet" (meat and fish), or, in general, for very easily digested food. Deficient bodily exercise and work is often an associated cause. Further factors consist in irregularity of life in every sense—in time of meals, of sleep, of work, and, of course, in the act of defecation itself.

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[*] Illoway: Constipation in Adults and Children, New York, 1897, is a very useful and complete monograph on constipation, which we can recommend to the reader who is interested in details.

As a secondary condition, habitual constipation is found in connection with a great variety of diseases belonging to every department of medicine and surgery, and no attempt will be made to enumerate them. Constipation, as a complication or sequel of other intestinal affections, will be treated of under the headings of these affections.

As to the other form of habitual constipation, that to which, in a strict sense, Nothnagel[1] applies this term, it is difficult to decide whether it is due, as he supposes, to a functional abnormality of the intestinal nervous apparatus; or, as Emminghaus[2] has recently announced on the basis of careful histological research, to changes in the splanchnics; or how far the condition is, according to Dunin[3], one of the features of anomalies of the central nervous system (neurasthenia, hysteria); or, finally, as Glénard[4] has endeavoured to show, whether it is dependent upon displacements of the intestines (enteroptosis).

It must be admitted that each of these hypotheses has a basis in clinical experience, but no single one will serve to explain the manifold varieties met with in daily practice. As Dunin has elucidated in his excellent paper, there is evidently in many cases a vicious circle, which when well developed may obscure the initial cause of the malady. For illustration, let us suppose a case such as we see almost every day. A woman who has previously been healthy begins to suffer from constipation and uses laxatives; gradually these lose their effect, and defecation becomes more and more difficult and less complete. Hand in hand with this goes a failure of nutrition, either as a result of the abuse of laxatives or from therapeutic measures (an "easily digested diet"), or as a result of the general failure of health or of anæmia or gastric disorders (atony, for example). The natural result is emaciation, prolapse of the viscera, and with it increase of the constipation, and finally, as the climax of all these symptoms, the clinical picture of well-marked neurasthenia.

Every one of experience will recognise that in this case the enteroptosis is not the cause but the result of the habitual constipation, and the same is true of the neurasthenia. On the other hand, emaciation from any cause may lead to prolapse of the viscera and thus cause constipation, or, more accurately speaking, favour it; and in this manner, as Dunin had in mind, pure neurasthenia may lay the foundation for the most obstinate kind of constipation.

Symptomatology and Diagnosis

Habitual constipation occurs in several grades of severity and in several clinical forms, the differentiation between which is of practical importance.

We may distinguish the mild, medium, and severe types. But what do we understand by this? That a certain accord exists between the duration and intensity of the disease is undeniable, and this would be a good diagnostic test if experience did not show that there are many exceptions. In patients who have become constipated from any cause, the condition may in a very short time run an obstinate course, and be very rebellious to treatment. Nevertheless, the duration of the disease, as well as the existence of an unmistakable hereditary or, perhaps more accurately, a family tendency which has shown itself in the development of the condition in early childhood, has a significance not to be underestimated.

As previously mentioned, I have found that we can judge of the intensity of the process by the results obtained from the use of purgatives. When the most severe drastics, used in large doses for years, fail to produce their effect, it is safe to assume that the case is a severe one, and, from a therapeutic standpoint, not a very promising one.

In badly neglected cases, especially in women, the fæcal accumulations may form tumours. These are sometimes situated in the large intestine, usually in the cæcum or the neighbourhood of the sigmoid flexure, and may cause the outline of the intestine to stand out in relief; or they may be located in the rectal pouch and attain such a size as to dilate the latter like an aneurismal sac. It is well known that fæcal tumours may exist and yet the defecation be apparently normal or even diarrhœal in character, a byway having been formed through which the dejections pass. Under certain unfavourable circumstances symptoms of obstruction which may require surgical intervention, may develop. These cases will be discussed in the chapter on Intestinal Stenosis.

Very convenient for clinical purposes is Fleiner's[5] division into two classes, the *atonic* and the *spastic* forms. My own experience would lead me to add a third, which might be called the *fragmentary*.

The *atonic* variety is the usual form of constipation that depends upon simple weakness of the intestine, such as usually develops under improper habits of living and eating. According to Fleiner, the stools are drier and firmer than usual, and consist of compressed and desiccated lumps or cylinders of large calibre, or of distinct particles or scybalæ bearing the impress of the sacculations of the colon.

The *spastic* form is due, according to Fleiner, to the retention of firm masses of fæces within segments of spastically contracted intestine, somewhat as in lead colic. It is found chiefly in neurasthenics, hypochondriacs, and in women with pelvic disorders. The stools have the following characteristics: long or short cylinders of small calibre, often no thicker than a pencil or the little finger, or spherical masses of fæces of the size of a hazel nut. The latter formation is not characteristic of spastic constipation, as it is also found in the atonic form; it is only when constantly present that it is significant.

I can confirm Fleiner's observation that there are numerous transition forms and combinations, and that both varieties may be associated with catarrh of the large intestine.

As I have said, my experience leads me to distinguish a third form, *fragmentary evacuation*, on account of the peculiar subjective and objective symptoms.

These patients have regular spontaneous movements of the bowels, but the evacuations are incomplete, and therefore the call to defecation is frequently repeated. They are obliged to go to stool every two to three hours, and each time, with great straining, pass small quantities of cylindrical, or pointed, spherical, or pulpy fæces. The patients may have a sense of pressure and tenesmus in the rectum, or complain of a feeling of fulness in the abdomen, so that they make renewed attempts to empty the bowels, which may result in the evacuation of more of such fragments, or be quite fruitless. This variety seems to be especially frequent in men, and depends, I believe, upon a sluggishness of the lower segments of the large intestine, or sometimes of the rectum alone. On palpation the latter may be found full of fæces shortly after one of these evacuations.

Although this form may merge into the others or be combined with them, yet I think it is entitled to separate recognition on account of its significant symptoms.

The following case is a good example of fragmentary stools:

Paul P., merchant, of Berlin, thirty-nine years old. Has been somewhat nervous ever since academic and university study. He suffers frequently from neuralgias, nervousness, and præcordial distress. When he takes physical and mental rest there is temporary improvement. At the present time he complains chiefly of intestinal symptoms. He has six to eight movements of the bowels daily. Each time the evacuations are small, and of either firm or pulpy consistence. Defecation is preceded by an uncontrollable tenesmus. No mucus or blood in the stools. The evacuations which occur during the night are especially troublesome. There is a marked sense of hunger after each passage. Examination of the intestines, particularly of the rectum and of the stools, shows nothing abnormal.

A prominent symptom of habitual constipation, particularly of the spastic form, is *intestinal colic.* Attacks of most violent abdominal pain occur with or without noticeable meteorism. They sometimes involve the entire abdomen, and sometimes only limited areas, occasionally lasting for hours, and subsiding suddenly after the expulsion of much gas or an evacuation of fæces. These colics are present not only in constipation, but also, though less frequently, when the bowels move regularly. Perhaps in the latter case the evacuations are incomplete. Nothnagel [*] has also called attention to this fact.

In habitual constipation Kobler[6] finds that albuminuria or cylindruria are not infrequently found; with the cessation of the intestinal symptoms they vanish.

The question already briefly discussed, as to the relationship between habitual constipation and certain cerebral manifestations—such as headache, sense of pressure in the head, psychic depression, which we designate at the present day by the comprehensive term neurasthenia—can be disposed of in a few words.

An unprejudiced consideration leads us to divide the patients into three groups: (1) Severe intestinal hypochondriacs, whose every thought and aspiration is centered upon the function of defecation; (2) neurasthenics, in whom the constipation is but one of many complaints; and (3) individuals who either have no nervous disorders, or complain of nothing more than a sense of pressure in the head, or pain, or mild general malaise. In my experience, the last-mentioned category includes the great majority of cases. This alone would show that clinical observation does not give support to the trend of the doctrine of auto-intoxication which was advanced by Vötsch[7] in the 70's, and during the last decade by

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[*] Loc. cit., p. 34.

Bouchard[8], Feyat[9], Glénard[4], and others. I may say that I have been able to permanently cure the constipation in a large number of neurasthenics and constipation-hypochondriacs, but the symptoms of neurasthenia do not vanish, and the attention is merely diverted to some other disturbance.

The *diagnosis* of habitual constipation seems at first sight to be an easy one. This is a great error. I feel bound to state that in the early years of my practice I used to make a number of mistakes in this direction. First of all, the rule must be laid down that when simple constipation is complained of a thorough general and local examination should never be omitted. Under the latter head rectal examination is certainly included, especially with reference to the presence of hemorrhoids, tumours, fissures of the anus or of the neighbouring integument, which, together with anomalies of the genital tract, hypertrophy of the prostate, tumours of the uterus or of the ovaries, retroflexions, etc., are often important etiological factors. In the General Section we have already spoken (page 76) of the frequent blunders made when fæcal tumours are present, and we have pointed out how these errors may be avoided. But aside from such mistakes, which can almost always be avoided by careful examination, there are severe affections of the intestines which may be concealed under the guise of simple habitual constipation. These are, first, stenoses of the intestine of benign character; secondly, stenosing intestinal carcinomata. These cases, obscure in their early stages, but later often revealed in a sudden and very disagreeable manner, will be described under their appropriate headings. The following remarks are limited to a few points of practical importance.

Individuals of or beyond middle age, who have previously been healthy and never have had any intestinal trouble, and who without appreciable cause begin to suffer from habitual constipation, should *a priori* be suspected of having intestinal stenosis (usually malignant). This suspicion is strengthened if there is progressive emaciation. The occasional occurrence of attacks of colic should attract immediate attention. In such cases, even in the absence of a tumour that can be felt, the chain of evidence is very nearly complete, and it only remains to recognise the objective signs of stenosis by careful clinical observation.

Next to these most frequent and dangerous mistakes, a permanent and severe grade of obstruction may be caused by adhesions between coils of the intestine, incomplete volvulus, chronic invagination, etc. It will suffice to mention them here, as they will be discussed in the chapter on Intestinal Stenosis.

It is undoubtedly of importance to distinguish between a constipation arising from insufficiency of the muscular coat of the intestine and that of catarrhal origin. These can usually be differentiated from each other by inflation and methodical filling of the intestine with water in one case, and by intestinal irrigation in the other. The methods by which these may be accomplished do not differ essentially from those described in the General Section and in the chapter on Enteritis.

TREATMENT

The therapeutic problem in habitual constipation is that of inducing regular and adequate evacuations of the bowels. It should not be considered solved until the bowels move regularly without the assistance of mechanical or medicinal co-operation, the diet being normal or nearly so. The ways in which this may be accomplished are numerous, and it seems to me that the energy and consistency with which any one of these is carried out counts for more in obtaining a good result than the particular method itself.

Sometimes, when one method or another fails, several methods may very often be combined, and we do not doubt that in this way also something may be accomplished. In everyday practice such a procedure may indeed be justifiable, for the physician takes what is of advantage from any source; but from a scientific standpoint I must utter a protest against superfluous confusion of therapeutic methods.

In the introductory chapter of his Guide to Clinical Therapeutics, Penzoldt[10] has very justly remarked that a combination of methods makes the estimation of the value of any one of them extremely difficult. When a good result has been obtained, it is not clear which has been the active or most active agent, and too much credit or discredit may be attributed to one or the other. Unsuccessful results may be due to neglect or omission of the essential feature while the patient is occupied with what is of no importance. Besides all this, a multiplicity of methods or therapeutic procedures, such as are observed in certain lay hygienic establishments as well as in some managed by physicians, is by no means necessary for the patient; he returns from them loaded with a confusion of false ideas, which are difficult to eradicate by the authoritative opinion of medical men. I think it proper to allude to this subject—which is equally pertinent in other disorders than chronic constipation—as a warning against the danger of the various physical or mechanical fads which have begun to mark the reaction against the abuse of prescription writing.

The methods for the treatment of chronic constipation which will be considered are the prophylactic, the dietetic, the mechanical, the electrical, the thermic, and the medicinal.

1. The *prophylactic* treatment of constipation, which I know to be too little appreciated, should begin in childhood, and consist in appropriate rules for the child. It is the mission and the duty of parents to supervise the intestinal activity of children, to teach them to have evacuations at a set time, and, when necessary, to modify the diet under medical advice until such movements are satisfactory. The habitual use of laxatives during the first few years must, as has already been said (page 190), be prohibited. Such measures are especially necessary in families which have an inherited tendency toward atony of the intestine. It is hard to exaggerate the importance of prophylaxis during pregnancy, a condition which experience teaches us is often, chiefly on account of the local conditions, associated with more or less constipation. In these cases it is to be combatted by appropriate food (see below), and, as von Wild[11] has urged in his excellent essay, by gymnastic exercises, so as to endeavour to increase the strength of the abdominal muscles. The same is true of pareses due to long rest in bed during infectious diseases or after operations, etc.

2. *The Diet.*—We have discussed the essential principles of a rational diet in chronic constipation in the General Section (see page 146 et seq.). These will suffice for the preparation of suitable special dietaries. Detailed and very appropriate diet schemes have been given by Penzoldt[12], Rosenheim[13], and Wegele[14]. We prefer that of Penzoldt, because it is very simple and practical:

7 A. M.—A glass of cold water.

8 A. M.—A liberal breakfast, with sweetened coffee, a good deal of butter, honey, and Graham bread or pumpernickel, after which the patient should go to stool.

1 P. M.—Midday meal of meat, a good deal of vegetables, salad, stewed fruits, farinaceous food, half a bottle of light wine (Moselle, or cider).

7 P. M.—Meat, with a good deal of butter; Graham bread, stewed fruit, and beer.

10 P. M.—Before retiring, fresh or stewed fruit.

The dietaries of Rosenheim and Wegele differ from the above in that they add, once or twice a day, 300 grams of buttermilk or kefir, which in severe cases increases the effect of the diet.

In the great majority of cases a permanently good result is obtained by the use of this method—that is, without systematic adherence to the above *régime* the bowels will move under the usual diet, provided it is rich in carbohydrates. Even in older, indeed in very chronic cases, contrary to my prognostication, I have seen very excellent results from the use of this simple constipation diet. Some cases soon have a relapse, partly because the patient is not persistent enough, and partly because—as in the case of medicinal agents—dietetic laxatives may lose their effect after a time. In the latter cases, which in my experience are certainly unusual, supplementary measures must be employed.

It may not be superfluous to remark that the diet above described is only suitable for uncomplicated cases of chronic constipation. When complications exist it can not be used, or must be materially modified. For example, this diet is obviously contra-indicated in diabetes mellitus, in obesity, or in a tendency thereto, and in well-developed alimentary glycosuria. The discomforts which it occasions (pyrosis, oppression, vomiting, pain, hemorrhages, etc.) in gastric atony, hyperacidity, ulcer of the stomach, gastric dilatation, carcinoma of the stomach, carcinoma of the intestine, etc., naturally will forbid its use in these conditions. Undue flatulence may contra-indicate such a large quantity of sweets and acids. A careful choice of the foods which are well tolerated, as shown by prudent variations and experiments, will accomplish the desired results. It would lead us too far to go into particulars with reference to all the considerations in question.

Everyday experience shows that the milder cases will get along with much less change in diet. For example, it will suffice in very many cases to give Penzoldt's advice as to the glass of cold water in the morning, especially if a little common salt is added; in other cases the taking of fresh or cooked fruit on an empty stomach or in the evening is sufficient; or the morning cigar may set up intestinal peristalsis. Nor do these exhaust the possibilities. We can occasionally utilize these facts in treatment, at the same time that they throw light upon the wide individual variations in the irritability of the intestines in different subjects, and on the necessity of taking this factor into account in each individual case.

3. *Mechanical Treatment.*—The most important of such meas-

ures is massage; it has already been discussed in the General Section (pages 170–172). To this may be added, in many cases, other mechanical therapeutic agents.

The simplest of these are exercise, and various systems of gymnastics.

The value of the former should not be underestimated, although experience teaches us not to expect too much from it. We often enough meet with chronic constipation in people such as farmers and officers who take active exercise. The fact that surprisingly good results are obtained in the treatment of constipation in spite of absolute rest in bed, shows that the importance of exercise has heretofore been very much overestimated. For these reasons I have been led to prescribe rest in bed, with suitable diet, in some cases of severe constipation; and I particularly remember the case of a lady who was suffering from a severe type of constipation—whose medical adviser, in his perplexity, finally felt compelled to order her to take a bottle of bitter water every hour—who was completely and permanently cured by four weeks of absolute rest in bed, with no other treatment than an appropriate diet.

Much more valuable than simple exercise are the various forms of indoor gymnastics, calisthenics, rowing, bicycling, riding, tennis, bowling, football, and the Swedish movements, when they are carried out more or less methodically. But they are not all equally useful. For example, my experience shows that bicycling does not exercise any especial influence over intestinal activity; indeed, one of my most obstinate cases was in the person of one who was moderately addicted to this sport. The same is also true of riding. Rowing seems to be more useful, but, unfortunately, it is not always available. For the same purpose, the so-called rowing machines have been extensively recommended by some writers.

The importance of calisthenics and systematic indoor gymnastics, as explained in numerous books on this subject (Schreber, Fromm, and others), should not be underestimated. One of the most useful movements for strengthening weak abdominal muscles consists in raising the trunk slowly from a horizontal to an upright position without the assistance of the arms or legs, and then allowing it to slowly drop back again; this should be repeated several times each day. It is best practised in a progressive fashion, very gradually increasing the angle at which the trunk is maintained by the muscular exertion. Ultimately the motion of rising may be made against a slight resistance, such as that of the hand of an assistant laid upon the forehead (von Wild). Another useful exercise is high kicking of the knee, so that the anterior surface of the thigh is brought into forcible contact with the abdomen.

As Williams[15], Lauder Brunton[16], and recently Ewald[17], have pointed out, the position of the body during the act of defecation, belongs in a certain degree to this class of measures. These authors emphasize the advantage of a squatting position, in which, as is evident, the abdominal muscles act upon the rectum to best advantage. Anyone who knows from experience how difficult it is to have an evacuation in a sitting posture with the legs extended (bedpan position), will concur in the advice of these authors, but, unfortunately, there are practical difficulties in the way of carrying it out.

4. Concerning the use of *electricity*, its indications and advantages, see the discussion on pages 172–175.

5. With the *thermic* measures may be classed the various forms of *hydrotherapy*, which have been described on page 158. Their effect depends chiefly upon an improvement in the general condition, by which, as we have seen, nutrition and intestinal activity are increased. Local or intestinal hydrotherapy is also of importance in the form of douches and enemata (page 177), the latter containing substances which soften fæces (oil, soap, glycerin, etc.). It is also known that the sudden local application of cold will increase intestinal peristalsis, and this fact may be taken advantage of in therapeutics. The most simple and at the same time a very efficient method, according to my experience, consists in the use of cold water compresses (of course without an impervious covering). A more powerful application is the use of cold, or alternate cold and hot jets or sprays over the abdomen (Scottish douche). The *ether* spray is another and very simple method, which I have used in obstinate cases for a number of years. Once or twice a day, for about five minutes, 100 cubic centimetres of sulphuric ether are sprayed upon the abdomen with the Richardson apparatus. The chilling so produced markedly stimulates the muscles of the abdominal wall, and presumably also the intestine, for it soon gives rise to an urgent desire to go to stool. I will merely select two of my case histories in which the ether spray gave brilliant results:

CASE I. *Obstinate constipation, not cured by diet alone. Complete cure by ether spray.*

B., resident of Berlin, fifty-four years old, has suffered for many years from severe pyrosis and constipation. Was operated on for hernia in 1896, but with little improvement in the constipation. Movements from the bowels occur only after laxatives or enemata. The urine contains a good deal of uric acid, but there are no other signs of gout. Examination of the gastric contents shows a high degree of hyperacidity with atony. Rectum empty. Irrigation does not show the presence of mucus. Constipation diet was first tried. A passage resulted each day, but only with great difficulty, and the amount was insufficient. In May, 1897, the ether douche was used twice daily for about five minutes. The evacuations were from this time on softer and more free. After a fortnight's use of the spray the stools were normal. Subsequently the patient used it from time to time when there was delayed evacuation. Since the year 1898 the bowels have been perfectly regular on simple constipation diet. He has gained 6 kilos in weight since the beginning of the treatment. The hyperacidity has been improved by the continuous use of citrate of soda.

CASE II. *Chronic constipation of three and a half years' standing. Great abuse of drastics. Results from diet unsatisfactory. Regular action of the bowels results immediately from the use of the ether spray.*

Miss L., of Frankfurt-am-Main, twenty-three years old. The patient has suffered for the past three and a half years from severe atonic constipation, with resultant anæmia and anorexia. Purgatives of the drastic order, which the patient has used for a long time, always cause severe pain. At one period membranous enteritis developed, but disappeared later. At first the treatment in my private clinic consisted in constipation diet, but the bowels would not move spontaneously. Evacuations only followed the use of enemata of soapsuds, glycerin, or oil. Fourteen days after admission the use of the ether spray was begun, for five minutes once daily. The bowels moved daily thereafter, and after ten days of this treatment the spray was left off. The patient gained 15 pounds in weight, and left after four weeks, the bowels being perfectly regular.

Such favourable results are not obtained in all cases, but, because of its simplicity in obstinate cases, the ether spray should be considered almost as important as the diet.

6. *Medicinal Treatment.*—In recent times there is a good deal of diversity of opinion among authors as to the benefit or the harm that may result from medicinal treatment—that is, from the use of laxatives. Even if we ignore the exaggerations of "the doctor who follows Nature," and whose stock in trade it is to denounce all drugs as poisonous and harmful, there still remains between leading clinicians and physicians a gulf difficult to bridge over.

As representing one set of opinions I may quote Dunin[3], who "most positively forbids the use of any sort of laxative whatever," and of the other, the elder clinician v. Liebermeister[18], who gives the advice that when regular stools can not be obtained without too much trouble by dietetic regulation, "suitable laxatives are to be used with regularity each day." Of the standard authors, Penzoldt[10], Nothnagel[1], Fleischer[19], Rosenheim[13], Ewald[17], and others take a middle position. We can especially recommend Penzoldt's thorough and clear presentation of the facts of the controversy.

In what follows I shall give the results of my own experience in this matter. I must concur in the opinion of other investigators who deny that laxatives always, or in a majority of cases, have a harmful action. I know numerous healthy people who have taken a daily laxative for ten years—particularly rhubarb—without any injury to the intestine or to their general health. These people would with justice indignantly refuse to exchange their efficient and simple pill for monotonous and burdensome dietetic regulations. From this sort of case—one which seldom comes under professional notice—are to be distinguished two other varieties: the first, in which laxatives do have an effect, but occasion gastric * or intestinal discomfort, loss of appetite, emaciation, etc., and second, those in which laxatives have either no action or only an inadequate one. In both cases it is not a question of the further use of laxatives, for by the time that the patients seek professional advice the milder agents have ceased to act, and they have found that the more powerful ones sooner or later become inert against the intestinal torpidity, or augment the other discomforts just mentioned.

It is in these very cases that a rational diet, possibly in conjunction with some of the above-described methods, achieves its greatest triumph; and, even when it is not fully successful—as sometimes happens in very protracted cases—a satisfactory result may be obtained with the assistance of small, perhaps minimal doses of some mild laxative, or, still better, by enemata of some suitable fluid (oil, etc.).

Thus the question as to the indications for the use of laxative drugs is virtually self-answered. [Aside from the above-mentioned cases in which their long-continued use has produced no injurious effects], we employ this class of drugs only when a constipation diet, patiently persevered in, and if necessary assisted by other methods, is found inefficient.

We have already expressed our views concerning the choice of laxatives in the General Section (page 186 et seq.), and have pointed out that each of them has its special use and indications. These are essentially dependent upon the state of the stomach and, in cer-

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* Wiczkowski has recently made the interesting observation (Archiv f. Verdauungskrankheiten, Bd. iv, S. 407) that laxatives materially diminish, while opiates increase, the acid secretion of the stomach.

tain cases, of the liver. For instance, calcined magnesia and Carlsbad salts are also very excellent antacid remedies, and podophyllin and euonymin equally good cholagogues. In appropriate cases advantage must be taken of such facts.

Laxatives may often be administered by the rectum in the form of small enemata or as suppositories. The most popular and efficient, especially in the milder cases, is glycerin in doses of 1 to 8 grams. It is evident that their effect does not extend beyond the lowermost segments of the colon. Hiller[20] and, more recently, Kohlstock[21] have recommended the use of the active principles of approved laxatives per rectum. According to Kohlstock, the most serviceable are aloin, cathartic acid, and, for especially obstinate cases, colocynthin and citrullin. The following are the formulæ:

℞	Aloin	1.0
	Formamid	10.0

(A suitable dose is 0.4 to 0.5 of aloin.)

℞	Colocynthin	1.0
	Spirit.,	
	Glycerin ... āā	12.0

(A suitable dose of colocynthin is 0.01 to 0.04.)

℞	Acid. cathartinic. e senna	3.0
	Aq. destillat	7.0
	Sod. bicarb. q. s. ad react. alkalin.	

(A suitable dose of cathartic acid is 0.6.)

℞	Citrullini	2.0
	Spirit.,	
	Glycerinæ ... āā	49.0

(A suitable dose of citrullin is 0.02.)

Kohlstock states that these concentrated enemata are prompt and painless in their action. The only obstacle to their more general use is their high cost.*

Up to the present time the subcutaneous use of purgatives (aloin, colocynthin, citrullin, etc.) has not been very successful. Reference should be made at this point to the subcutaneous use of magnesium sulphate in doses of 0.12 to 0.18, as recommended by Wood[22] and by Eakins[23]. Wood states that he has obtained results in 70 per cent of cases of constipation; and Eakins, that even in a case of fæcal obstruction he obtained copious evacuations and cessation of the threatening symptoms after ten hours. Scarbinato[24] also had positive results, although, as he mentions, the effect of subcutaneous treatment is neither marked nor constant.

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* The preparations mentioned are manufactured by Merck (Darmstadt).

My own investigations with doses as large as 0.5 of magnesium sulphate were not productive of any distinctly noticeable laxative effect.

In the same communication Scarbinato describes another procedure which is scarcely known, at least in Germany: the endermic treatment of constipation by croton oil (6 to 10 drops in 15 to 20 gm. ol. olivæ). In four patients with chronic constipation, this procedure produced regular fluid movements, often accompanied by abdominal pain. Oleum ricini, used in the same way, gave negative results.

Those substances which set up peristalsis by mechanical irritation occupy a mid-position between medicinal and dietetic agents. Among these are the preparations of linseed, of which a tablespoonful is allowed to soak in water and taken as a drink on an empty stomach. A particularly agreeable and, as I know, an efficient variety is the linseed of Tarin, which is distinguished by its large size and elegant shape. The large amount of oil contained in these preparations seems to account for part of the effect. It appears to me very possible for some of the seed husks to gain access to the vermiform appendix and thus give rise to inflammation. This may be a purely theoretical idea, but it has nevertheless deterred me from a very extensive use of this simple and efficient remedy.

7. The *hydrotherapeutic* treatment of chronic constipation has been discussed in the General Section (page 158).

Among the complications of chronic constipation, the treatment of flatulent colic deserves a brief discussion.

As we are here dealing with a spastic condition of the intestinal canal, the preference should be given to opium in small doses (ext. opii, 0.01 to 0.02). Such doses will not only allay the pain but will promote an evacuation. The use of laxatives, or, better still, of a suitable enema, is indicated only after the painful contractions have subsided.

B. DISPLACEMENTS OF THE INTESTINES

Symptomatology and Diagnosis

A short review of the most important anomalies of position of the intestines has been given in the General Section (page 20 et seq.), and to this we refer the reader. In what follows, an attempt will be made to discuss briefly the clinical manifestations and the resultant therapeutic indications. For practical purposes, dislocations of the large intestine need be alone considered. Those of the small intestine produce appreciable symptoms only when there is a marked interference with peristalsis, such as may arise from acute kinkings, as caused by tumours, adhesions, compressions, etc. We shall return to this subject in the chapter on Stenoses of the Intestine. Malpositions of the large intestine may either exist for a long time without causing any disturbance of well-being, or may occasion manifold symptoms, or, finally, may disguise the clinical picture to such a degree that only a lucky chance, or an autopsy *in vivo*, or sometimes only a post-mortem examination, will show the true condition of affairs.

The functional disturbances which, as has been said, are sometimes present were not unknown to the older physicians—Morgagni, De Häen, Esquirol, and Ruysch. Virchow[25], in his famous treatise on the Diseases of the Abdomen, from an Historical, Critical, and Scientific Standpoint, has long ago and in a classical manner described the importance of peritoneal fixation as affecting the onward progress of fæces. The pathological importance of displacements was at times quite forgotten or underestimated, and at times unduly exaggerated (Esquirol, Vötsch), until recently the subject has been given a new prominence by Landau's fruitful researches on the subject of floating kidney and pendulous abdomen, and Glenard's original though somewhat fantastic doctrine of enteroptosis. The valuable contributions of Leichtenstern[26], Curschmann[27], and Fleiner[28] have notably enriched our knowledge concerning the origin and clinical significance of displacements of the intestines.

According to Fleiner, displacements of the colon may owe their origin to abnormal curves and angular flexures which interfere with the fæcal movements. These are favored by increased pressure upon certain segments of the large intestine by ill-fitting corsets, belts, or, in the case of men, by an habitual stooping carriage and sedentary mode of life. In this way dilatation of the affected segments occurs, fæces and gas accumulate, and the clinical picture of atonic constipation is developed.

Soon organic changes in the wall of the intestine occur. Catarrh develops, and there is then added diarrhœa, or diarrhœa alternating with constipation, or constipation with membranous enteritis. Neuralgic colicky pains set in, which, according to Fleiner, are easily mistaken for intercostal neuralgia, biliary colic, renal colic, spinal crises—even for duodenal ulcer. Fleiner has given the details of several very instructive clinical histories which show how difficult it is to avoid mistakes, especially in confounding these paroxysmal pains with biliary colic. I can add from my personal experience that it frequently happens, especially in women, that the symptoms of gastrointestinal neurasthenia are present. There are anorexia, oppression in the epigastrium, intestinal colic, flatulence, constipation, mental depression, disinclination for work, loss of weight, etc. Objective signs are ptosis of the stomach and colon, displacement of the kidneys, liver, or spleen, and sometimes of the uterus.

Many physicians are puzzled when they come to deal with this condition. Some think of anæmia and prescribe iron; others suspect gastric or intestinal catarrh and prescribe bismuth or laxatives; others diagnosticate hysteria and attempt to cure with valerian, bromids, or asafœtida.

Displacements of the intestine may, however, cause very dangerous changes in the intestines, and may endanger life. Thus Curschmann has described two cases of upward flexure of the cæcum with absolute occlusion of the intestine from the acute bending. Both patients died with symptoms of acute obstruction. When there are abnormal bends of the transverse colon and its flexures, there may be an acute angle formed which causes partial or total obstruction.

These changes of position are of clinical importance also, because they may lead to errors in diagnosis not liable to arise in any other way. Curschmann has reported very interesting cases in which the cæcum was bent so as to bring the vermiform process directly into contact with the liver (see Fig. 24). If the appendix became inflamed in this situation the exudate would be close to the right costal arch.

In case of congenital shortness or absence of the ascending portion of the colon, the cæcum with its appendix might be close to or behind the liver. In a case described by Curschmann (see Fig. 25), a perforating perityphlitis was present while the clinical signs were those of cholelithiasis.

Dislocations of the colon are especially liable to be confounded with diseases of the liver. von Leube[29], and subsequently Fleiner[28] and Curschmann[27], have called attention to the fact that abnormal elevation and gaseous distention of the transverse colon may reduce or abolish the area of liver dulness.

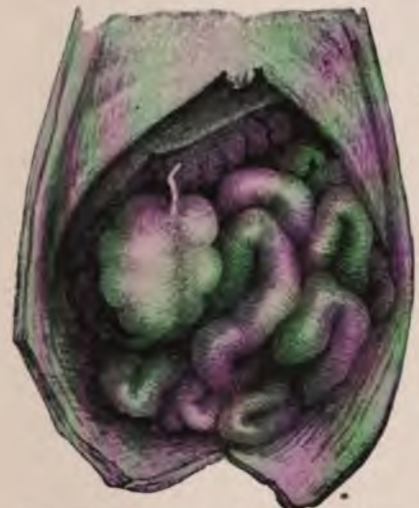

Fig. 24.*—Vermiform Appendix in Contact with the Under Surface of the Liver. (Curschmann.)

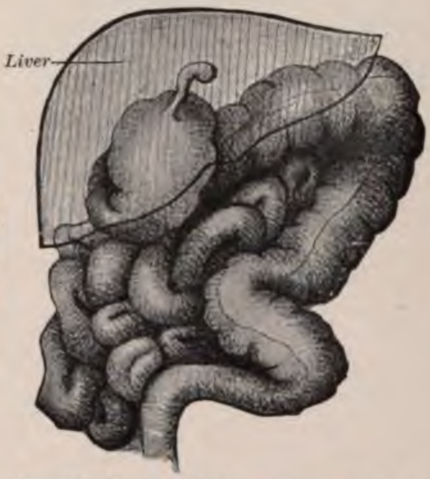

Fig. 25.—Vermiform Appendix lying behind the Right Lobe of the Liver. (Curschmann.)

I have often observed the absence of liver dulness in males. As Curschmann observes, this may often cause difficulties in mapping out the edge of the liver in hepatic cirrhosis. The normal position of the liver dulness in the anterior and posterior axillary lines enables a decision to be reached. In one of the cases which Curschmann has described and illustrated, a duodenal ulcer was mistaken for a subphrenic abscess because the flexures of the colon were absent, both limbs running almost parallel. According to Curschmann, when the flexures are exaggerated into loops the splenic dulness may be obscured.

It is well known that loops with coincident elongation are found with especial frequency in the transverse colon and at the flexures. They are chiefly single; next in frequency come the **M** or **V** shapes, and rarely the double looping seen in Fig. 26. If the flexures become still further enlarged, they may dip down as far as the brim of the pelvis. It has long been known that abnormal loops of the sigmoid flexure may give rise to serious mistakes. We have already mentioned (page 23) that when the sigmoid flexure is strongly inflated, it almost always encroaches upon the right lower quadrant of the abdomen, and therefore lies close to the cæcum. When there is a suspicion of volvulus in that region, the physical signs of distention of the sigmoid flexure should be looked for. The importance of abnormal loops of the sigmoid flexure when operative procedures are in question, when an artificial anus is to be constructed, or when a loop is to be exsected, is of course evident. The difficulties may be very much increased when an abnormally long sigmoid flexure is arranged in a double (Fig. 27) or in multiple loops (Fig. 28).

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* Thanks are due to Professor Curschmann, of Leipzig, for permission to reproduce Figs. 24–28.

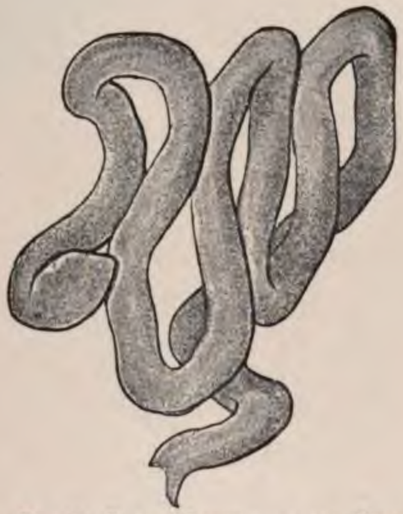

Fig. 26.—Double Looping of the Transverse Colon. (Curschmann.)

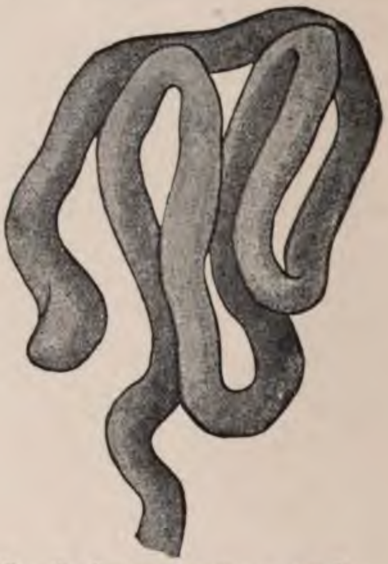

Fig. 27.—Double Looping of the Sigmoid Flexure. (Curschmann.)

The latter condition may be considered on the border line between normal and pathological; it presents favourable conditions for the formation of a volvulus.

The *diagnosis* of certain forms of intestinal dislocation, such as the common depression or elevation of the transverse colon, offers no special difficulty. In most instances gross variations of this kind can be determined with probability or certainty by inflation of the intestines with air, by methodical distention with water, or by careful percussion. The case is quite different with displacements of the cæcum, deformities and dislocations of the sigmoid flexure, absence of the flexures, abnormal looping, and other irregular conditions. The possibility of an anomaly of position must be taken into consideration in making a diagnosis. Curschmann was fortunate enough to do this in one instance. Perhaps the capsule method, with the assistance of the Röntgen rays, as recently described by Levy-Dorn and myself[30], will have some value for the recognition of these anomalies of position or form. Practical experience is, however, still lacking. When the clinical signs are obscure, the possibility of such variations must be taken into consideration.

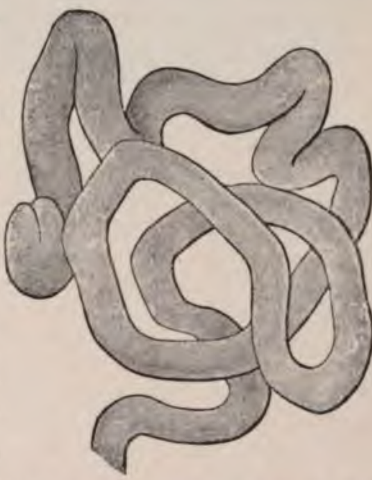

FIG. 28.—MULTIPLE LOOPING OF THE SIGMOID FLEXURE. (Curschmann.)

TREATMENT

The description of the treatment of changes in position or form of the bowel touches upon many chapters of intestinal pathology; it is not possible, therefore, to enter into details in this place. Prolapse of the transverse colon, however, which is usually only a part of a general visceral ptosis, requires a few brief remarks. The fundamental therapeutic maxims agree essentially with those in gastroptosis (see Diseases of the Stomach, Part II, page 183). They consist primarily in complete rest in a horizontal position and in a strengthening diet. The latter should not be a routine one, such as Weir-Mitchell's, but should be adapted to the necessities of each case, with reference to the functional disturbances of the alimentary canal.

Constipation or colitis must be treated in the manner advised in the chapters on these affections. By the use of massage, hydrotherapy, and electrotherapy, the effects of the dietetic prescriptions may be enhanced and the general condition improved. A suitable abdominal supporter is useful in assisting the weak abdominal muscles.

Treatment in a sanitarium is far preferable to home or ambulatory treatment.

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CHAPTER XV

ULCERS OF THE INTESTINES

Introductory Remarks.—An extraordinary variety of ulcers occur in the intestinal canal. It is unnecessary to describe them all in this chapter, as many are only complications or localizations of diseases which do not fall within the province of this work. Such, for example, are the ulcerations accompanying acute infectious diseases (typhoid fever, acute dysentery, diphtheria, anthrax, sepsis, erysipelas, variola, puerperal fever, leprosy), as well as constitutional diseases (gout, scurvy, leucæmia). The toxic ulcerations from mercury, arsenic, antimony, as well as the so-called uræmic ulcers, bear such slight relations to intestinal pathology that there is no necessity for describing them here. There remain only those forms of ulceration which, from their clinical symptoms, pursue an independent course. These are, naming them in the order of their frequency: *the catarrhal ulcer, the follicular ulcer, the stercoral ulcer, the tuberculous ulcer, the chronic dysenteric ulcer, the syphilitic ulcer, the amyloid ulcer, and finally the embolic and thrombotic ulcer.* The duodenal ulcer, a peculiar clinical type, will be described separately, and in that connection a few remarks will be made on ulcers due to burns.

Catarrhal and *follicular* ulcers are tolerably often observed in intestinal catarrhs. Their favorite location is the large intestine, and it is only exceptionally that they are found higher up. They owe their origin to slight losses of epithelium, which permit of the entrance of organisms which excite inflammation, or of substances which are chemical irritants. The superficial strata of the mucous membrane break down and a superficial erosion develops; if the process goes on, an ulcer of more or less depth may be formed which may penetrate to the serous coat, and terminate in perforation. Several small ulcers may become confluent and give rise to a single large one. If the ulcer heals, subsequent cicatricial contraction may lead to intestinal stenosis.

The follicular ulcers originate primarily from an inflammation of a follicle (suppurative follicular enteritis), in which the swelling gradually increases, suppuration and rupture occur, with resulting loss of substance—the follicular ulcer. Sometimes these points are so numerous that the mucous membrane presents a sievelike appearance. From undermining of the mucous membrane, these ulcers, as in the variety just mentioned, may coalesce to form larger ones of sinuous form.

The *stercoral* ulcer, or the *decubital* ulcer of Grawitz, is found almost exclusively in the large intestine, and especially at those points at which the pressure of the fæces is most marked—at the flexures of the colon, in the cæcum, the sigmoid flexure, the rectum, and very frequently in the vermiform appendix. In the latter situation it may, under certain circumstances, give rise to perityphlitis. Stercoral ulcers are frequently seen to develop above stenosed portions of the intestine. They may be superficial or deep, and lead to extensive loss of substance with suppuration. If they heal, stenosis may follow from cicatricial contraction, but extensive strictures secondary to stercoral ulcers are of great rarity.

The *tuberculous* ulcer is by far the most important variety, and the one which has been most carefully studied. A distinction is made between primary intestinal tuberculosis, which develops in the intestine of an individual who has heretofore been free from tuberculosis, and secondary intestinal tuberculosis, which arises in connection with some other tuberculous affection. The occurence of primary intestinal tuberculosis is still disputed by Klebs[1] and von Leube[2]; but at the present day we must admit that unimpeachable observations (Behrens[3], Eisenhart[4], Wyss[5], Melchior[6]) have demonstrated that it may rarely occur in older children and adults. In earliest childhood intestinal and mesenteric tuberculosis is of very frequent occurrence. Investigations, particularly those of Bollinger and his pupils, give convincing evidence that this form of tuberculosis should be regarded as dietary (milk and the flesh of tuberculous cows).

Secondary intestinal tuberculosis, on the other hand, is one of the most frequent complications of pulmonary tuberculosis. According to the statistics of Eisenhart[4], based upon 1,000 autopsies, it was present in 56.3 per cent. Other authors, such as Hamann[7], find the percentage to be higher; and Herxheimer[8] states that in 58 cases there was only 1 in which tuberculous disease of the intestine could not be found. The mode of origin of intestinal tuberculosis, long ago attributed by Klebs to the swallowing of tuberculous sputum, is now recognised to be an auto-intoxication with material containing bacilli. This may even be proved in an indirect way from the statistics of Eisenhart. In them it appears that out of the 1,000 autopsies on [all varieties of] tuberculous patients, of whom 567 were cases of intestinal tuberculosis, there were only 3 cases in which the intestinal tuberculosis was not associated with pulmonary tuberculosis. Infection of the intestine by the tubercle bacillus naturally results if erosions are present; the researches of Orth[9] are especially instructive on this point. But it is important to note that, according to the investigations of Fischer[10], Dobroklonsky[11], Tschitschowisch[12], and others, tuberculosis may be conveyed to the mucous membrane of the intestine even when its epithelium is intact.

Tuberculous lesions are not distributed uniformly throughout the intestines; some regions—the lower part of the ileum and of the cæcum—show a special liability to invasion. The very slow movement of the chyle in these regions is the chief cause why this is the site of preference. Below the cæcum and above the ileum the development of tuberculous ulcers is much more infrequent. The process usually begins in Peyer's patches and in the solitary follicles. Small nodules are developed in them (miliary tubercles) and the lymph follicles become smaller. By their rupture ulcers are formed. Fresh eruptions of tubercle spring up from its base and in its neighbourhood. The original lenticular ulcers increase in size by coalescence, and, breaking through the muscular coat, reach the serous covering and sometimes penetrate into the abdominal cavity. The ulcers may lie with their longer axis parallel to the course of the intestine (longitudinal), or at right angles to it (encircling). The former develop in a Peyer's patch, the latter follow the course of the vessels; yet some have quite an irregular outline. Tuberculous ulcers of the intestine exhibit in the main but a very slight tendency to heal. Out of Eisenhart's[4] 567 cases, only 10 showed ulcers which had completely healed; in 25 cases there was partial cicatrization. In consequence of cicatricial contraction, intestinal tuberculosis may produce simple or multiple stricture, in rare cases complete occlusion[13]. In the chapter on Intestinal Stenosis this subject will be taken up more in detail. Perforation of a tuberculous ulcer is a rare occurrence (about 5 to 10 per cent). It usually takes place in the cæcum or vermiform appendix, and opens into a space shut off by previous inflammatory adhesions, and only very rarely into the general peritoneal cavity.

A few remarks on ileo-cæcal tuberculous tumours may be introduced at this point, because they arise from tuberculous ulcerations, although, strictly speaking, they fall under the head of tumours.

Our knowledge of ileo-cæcal tumours is of recent date, and the credit for it is chiefly due to the experience gained from the operative surgery of the intestines. Following Conrath[14], Durante (1890) was the first to point out the features of resemblance and the differences between carcinoma and tuberculous tumour of the cæcum. In 1891, he was followed by Billroth[15], Henri Hartmann and Pilliet[16], and Salzer[17], who laid stress upon the tuberculous character of the tumours in question. The excellent work of Czerny[18], König[19], Körte[20], Hofmeister[21], and Conrath[14] has so far advanced our knowledge of the pathology and operative treatment of tuberculous tumours of the ileo-cæcal region that at the present day our clinical knowledge of them is complete in all essential details. From the medical side, however, they have received very little attention except in the paper of Obrastzow which will be alluded to later, so that I think a detailed consideration of the subject is indicated in this place.

The tuberculous tumour (see Fig. 29) is usually the product of inflammatory infiltration from multiple tuberculous ulcerations. These partially cicatrize, with the formation of a large amount of scar tissue, which gradually contracts so as to cause a stenosis of the lumen of the intestine. It is evident that such stenosis will favour hypertrophy of the coats of the intestine. The contraction is most marked in the vicinity of the ileo-cæcal valve because the tissue shrinkage is greatest at this point; the valve itself is usually involved in the process.

The ulceration may originate in the serous coat and extend deeply. Conrath attributes this to a direct infection from localized tubercular disease of the lymph glands; while the mucous-membrane form is considered to be an auto-infection from tuberculous sputum, or as a primary tuberculosis—a tuberculosis from ingesta in the stricter sense. Conrath traces the fact that cæcal tuberculosis usually remains localized, to several causes. One is, that the tubercular deposits in the subserous layer do not contain so many bacilli as those in the deeper strata, and thus generalization is hindered. For this reason the pulmonary phthisis observed in connection with cæcal tuberculosis is usually of mild form, and in contra-distinction to fully developed phthisis, the opportunity for bacillary infection of the intestine is relatively small.

Evidently we must not, even in this case, lose sight of the fact that on account of its location the cæcum affords a favourable seat for the deposit of tuberculous products, and most unfavourable conditions for their cure. The conditions are apparently very analogous to those in primary tuberculosis of the appendix, which once estab-

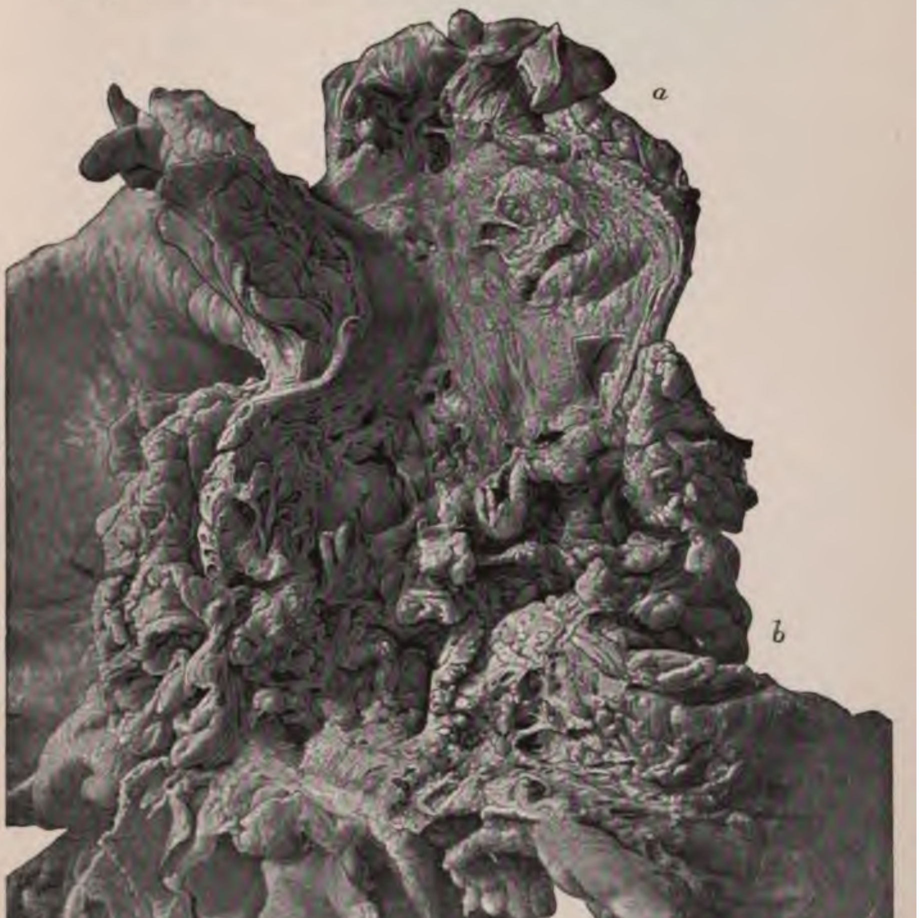

FIG. 29.—TUBERCULOSIS OF THE CÆCUM. (WÖLFLER—CONRATH.)
a, junction of cæcum and ascending colon; *b*, junction of ileum and cæcum.

lished, in like manner and doubtless for the same reasons, and in spite of any sort of treatment, leads to progressive changes.

The *chronic dysenteric* ulcer develops as a sequel of the acute form. The symptoms may be those of an unusual prolongation of the disease, or they may set in shortly after apparent cure by one or more relapses; or, finally, what was originally a catarrhal diarrhœa may terminate in dysentery. In its essential features the anatomical picture of chronic dysentery resembles that of the acute form, and is characterized by the formation of a variable number of deep-seated ulcers of the large intestine with raised and undermined edges. Accompanying this there are the symptoms of an intense catarrh of the large intestine. If the ulcers heal, the intervening islets of mucous membrane become so much more prominent that they may resemble true polypi. Contraction may lead to stenosis, but experience shows that this is not of frequent occurrence. It only exceptionally happens that dysenteric ulcers perforate, for the serous coat over them is usually thickened by inflammation. In less pronounced cases the intestine shows only the signs of a severe catarrh with swelling or suppuration of the follicles, or the formation of simple catarrhal ulcers. Between these there exist a great variety of transition forms which sometimes follow the type of catarrhal enteritis, and sometimes of true dysentery.

Syphilitic ulcers are extremely rare in the small intestine (they are most frequent in the newborn); they are more common, but still rare, in the large intestine; they are most common in the rectum. Syphilitic ulcers of the large intestine are usually formed by the breaking down of gummata in the mucous or submucous coats. They begin as superficial bulbous elevations, which break down slowly and leave ulcers characterized by sharp borders and a yellowish, flocculent, grayish-white base. Under certain conditions they may coalesce, with extensive loss of substance, the tendency being to superficial rather than deep ulceration. Perforation into the abdominal cavity of a syphilitic ulcer of the large intestine has not, as far as I know, been described, but perforation into some neighbouring organ, especially from the rectum, is not at all infrequent. Stenosis of the large intestine from syphilis is very rare. For syphilitic ulceration of the rectum, see the chapter on Diseases of the Rectum.

Amyloid ulcers, according to some authors, are rare, and according to others (Colberg, Courtois-Suffit, etc.) they are frequent. The pathological anatomists (Orth, Ziegler, Birch-Hirschfeld) speak with great reserve concerning the occurrence of this form of ulcer. At the present time they have no clinical importance.

Embolic or *thrombotic* ulcers arise from the occlusion of small twigs of the mesenteric artery, as a result of endocarditis or of atheroma of the large vessels. A small hemorrhagic infarct results from the embolism, and is followed by necrosis and ulceration. The ulcers are located chiefly in the small intestine, from the duodenum down to the cæcum; below this point they are very seldom met with. The ulcers are of various sizes, depending upon the extent of the infarction. Sometimes the ulceration is very considerable, extending through the entire thickness of the intestinal wall, and perhaps leading to perforation into the abdominal cavity. Septic emboli from ulcerative endocarditis may cause either small hemorrhages or very minute embolic abscesses between the mucous and submucous layers. These rupture into the interior of the intestine, and give rise to multiple ulcerations.

SYMPTOMATOLOGY OF INTESTINAL ULCER

The symptoms of intestinal ulcers are very varied, and depend not only upon the kind, but upon the localization, the number, and the extent of the ulcerative processes. It must be kept in mind that, as a rule, a more or less intense catarrh accompanies every form of ulceration. The changes in the stools thus produced (constipation, diarrhœa, mucous stools, bloody stools) in turn favour the progress of the ulceration and hinder cicatrization. We have therefore to consider a variety of conditions which, to a very considerable degree, must influence and modify the clinical picture of intestinal ulceration. In the first place, there are no distinctive signs for the differentiation of the various kinds of ulcers. Even the recognition of tubercle bacilli in the stools—to repeat what we have already emphasized (page 120)—has only a very limited significance. For this reason a separate symptomatology of the various forms will be omitted, and the discussion will be limited to the characteristics which they possess in common.

It is undoubtedly true that ulceration frequently occurs without any symptoms whatever. Every physician who has been present at many autopsies in cases of phthisis has observed intestinal ulceration, sometimes of considerable extent, which had caused no appreciable symptoms during life. This is also true of amyloid degeneration of the intestine with ulceration, and especially so for stercoral ulcers, as well as for the catarrhal and follicular forms. It is difficult to determine whether there may not have been slight subjective symptoms (constipation), and changes in the stools (admixture with blood, mucus, or pus). The clinical history gives us little positive information. In the majority of cases of ulceration, however, there are symptoms which permit the diagnosis to be made in some cases with likelihood, and in others with certainty.

We shall next describe:

(*a*) *The Subjective Symptoms.*—The most important symptom is *pain.* As has just been mentioned, pain may be entirely absent in ulcer of the intestine. When it is present, it does not give us any clew to the variety or location of the ulcerative process. Though the patient be intelligent, his statements as to the subjective sensations of pain are very vague, and it is only exceptionally that they serve to indicate the location of the process. The objective sensitiveness on pressure is more valuable. This tenderness seems to me to be most marked in severe forms of intestinal tuberculosis, and in some cases I have found it localized and very persistent in the region of the umbilicus. It has to be distinguished from the tenderness of chronic dysentery, which is more diffuse and extends over the descending colon and sigmoid flexure. In my experience this too may be absent. The intensity of the pain is of some importance in estimating the extent of the ulcer and the progress which it is making toward the external surface of the intestine. In cases of very decided tenderness deep ulceration may with circumspection be thought of.

(*b*) *The Objective Symptoms.*—Of these, the nature and condition of the evacuations from the bowels are of chief importance.

In cases of well-marked ulceration of the bowel the passages may be normal, or there may be constipation. It is important to remember this in passing judgment on individual cases.

The younger Frerichs describes two very instructive cases in his Contributions to the Study of Tuberculosis (1882). They were both cases of pulmonary consumption. One of them had a profuse diarrhœa with elevation of temperature, so that a diagnosis of typhoid was made. The other patient was persistently and obstinately constipated. In both cases the autopsies showed that the cause of the bowel symptoms was a widespread intestinal tuberculosis. In the second case, in addition to the tuberculosis of the ileum, there was extensive tuberculous ulceration of the colon.

Nothnagel advances a plausible hypothesis to account for these cases, viz., that the destructive process has either completely destroyed the nerves in the base of the ulcer, or that the continuous irritation has exhausted their sensitiveness to the usual stimuli. The stools usually show marked deviations from the normal; there is diarrhœa, or diarrhœa alternating with constipation. It is very probable that the seat of the ulceration has an influence in determining which of these conditions will predominate, since experience has shown that diarrhœa is less frequent when the ulceration occurs high up than when it is below the ileum. As has already been said, the existence of a simultaneous enteritis has an extremely important, perhaps the most important, influence. For instance, there is scarcely ever any severe diarrhœa in ulcer of the duodenum, because the process is a localized one, and therefore the catarrhal condition is limited to a very small portion of the intestine.

The presence of abnormal constituents, such as blood, pus, and shreds of necrotic tissue, is more important than the consistence of the stools. *Blood* may be present in various forms: as fresh or decomposed blood, or intimately mingled with the dejections, yet recognisable by the eye, or, finally, only to be detected by the microscope. The old maxim that blood from the upper part of the intestinal canal is materially altered in appearance when voided is true, as a rule, in ulceration of the intestine. But, as every one knows from the pathology of typhoid ulcer, blood which is unchanged may come from the subdivisions of the small intestine, provided it is quickly expelled. Blood is always passed in an unchanged condition when it comes from the lower part of the small intestine or from the colon, and, according to the amount of the hemorrhage, appears as an enterorrhagia, or is intimately mingled with the dejections. Smaller hemorrhages are usually easily recognised in the same way; minute ones only by microscopic, chemical, or spectroscopic methods.

Hemorrhages are not, however, a necessary symptom of intestinal ulcer. In dysenteric and typhoid ulcers they are very frequent, and in tuberculous and catarrhal ulcers relatively infrequent. In tuberculosis of the intestine, according to Girode[22], the stools often have a dark colour, similar to the coffee-ground vomit of gastric cancer; and this he attributes to repeated oozing of blood from the ulcerations. I have observed the same appearances, but they do not afford conclusive proof of the presence of blood.

Admixture of *pus* in the fæces is a very important symptom of intestinal ulcer. However, the finding of pus is not absolutely conclusive, since it may come from some abscess in the neighbourhood which has ruptured into the bowel, or may occur in croupous or dysenteric conditions, or from ulcerating tumours of the bowel. For this reason, as I know from personal experience, the diagnosis may become extremely difficult. It is very important to ascertain whether the pus is voided pure or mixed with blood. In the former case it points to the presence of an abscess adjacent to the intestine, while in the latter it indicates that the pus has originated in the bowel itself. Aside from this consideration, it may be stated that the presence of pus makes intestinal ulceration in the highest degree probable. On the other hand, its absence does not negative the existence of ulceration.

Like the gastric contents in ulcerating carcinoma, the passages acquire a penetrating fetid odour when mixed with large quantities of pus. This differs so characteristically from the normal odour of the fæces that it can scarcely be forgotten by one who has ever appreciated it. The importance of this sign lies in the fact that purulent stools may often be detected in this way when a formal inspection has been neglected. In many cases the unaided eye suffices to determine the presence of pus. Small quantities of pus can only be recognised by the microscope; but, if the fæces are spread out on a black dish, yellowish-green specks, of the size of the smallest millet seeds, will sometimes be seen and recognised as pus.

Fragments of intestinal tissue, when present, are always the result of necrotic processes, and are found only in acute and subacute dysentery. It has often been asserted that the presence of a formation resembling frog spawn or sago grains is characteristic of intestinal ulceration. But there is now no doubt that Virchow was right when he declared that these are vegetable products, which may be found in the stools in a great variety of conditions. Of the intestinal bacteria, the tubercle bacillus is the only one which has any, and even that a very limited, significance. In the General Section we have discussed the diagnostic value of the presence of the tubercle bacilli, and it is sufficient to repeat that it is only their continuous absence from the sputum and constant presence in large numbers in the stools which justifies any positive conclusion. In tuberculosis of the rectum a doubtful diagnosis may be made certain by the removal of material containing bacilli from the ulcer itself.

(*c*) *Meteorism.*—This may accompany intestinal ulceration, and is seen fairly often in tuberculosis of the bowel and in dysentery. In other forms of ulceration I have not found meteorism a constant symptom, unless there existed also some narrowing of the lumen of the bowel. In the above-mentioned varieties (e. g., tubercular and dysenteric ulceration) meteorism may, of course, be considered a sign of intestinal paresis.

(*d*) *Fever.*—Fever is not present in simple ulceration of the intestine—i. e., in the catarrhal or follicular forms. But irregular fever is a very important clinical symptom of dysenteric and tubercular ulcers. Sloughing carcinomata, as has been mentioned in the chapter on that subject, may cause an irregular and sometimes very marked pyrexia; the same is true of para-intestinal abscesses.

(*e*) *The Urine.*—Up to the present time the examination of the urine has not been of any special importance. The interesting relation that exists between ulceration of the intestine and albumosuria after the administration of peptone in tubercular ulceration of the intestine (see page 135) is worthy of further investigation. Under some circumstances, the Ehrlich diazo reaction may be of service in differential diagnosis (see p. 276).

(*f*) The *general health* may suffer markedly from the fever, hemorrhages, and suppuration, as well as from the diarrhœa. I have, however, seen patients who, in spite of frequent suppurations, remained well nourished. That this feature depends chiefly on the primary cause needs no explanation.

The symptomatology of *ileo-cæcal tumours* may be introduced in this place.

The age and sex are of importance. According to Conrath's collection of 85 cases, the frequency of tuberculosis of the cæcum is pretty nearly the same in both sexes. Much more than one half (65 per cent) of the cases were between the ages of twenty and forty years. Very few were found in the fifth and sixth decades. From Conrath's statistics there is a significant preponderance of females between the ages of twenty and thirty, while in those between thirty and forty the proportion of males is markedly greater. Cæcal tuberculosis usually begins insidiously, or has no characteristic symptoms. There may, for example, be constipation, alternating perhaps with diarrhœa, but there is nothing to indicate that an incurable destructive process has begun. There is no change in the picture until the characteristic phenomena of stenosis or a tumour become manifest. The distinctive signs of chronic stenosis of the bowel develop—the occasional attacks of colic with nausea or vomiting, the visible tetanic contractions of the intestine, the constipation persistent in spite of the usual remedies, and, as a result, a severe loss of nutrition. In prolonged cases the marasmus is as marked as the cachexia of cancer. Occasionally there may be hemorrhages from the bowel, or blood may be mixed with the stools. As the disease is usually associated with a manifest or incipient pulmonary phthisis, irregular fluctuations of temperature are frequently observed.

The most important symptom of cæcal tuberculosis is the tumour. Its size varies; it is usually made more accessible to palpation by filling the rectum with water. At first the tumour is more or less movable, but later it may become quite fixed by adhesions or thickening of the mesentery. Under such conditions it may lie immediately under the abdominal wall, and thus lead to serious mistakes in diagnosis. Furthermore, it may suppurate, break down, and rupture externally, forming an artificial anus; or the abscess may perforate into the abdominal cavity, or into one of the neighbouring organs.

The course of cæcal tuberculosis is usually slow. Many cases lasting two or three years have been observed. As the tumour doubtless has a long period of latency, the beginning of the disease evidently dates much farther back.

Under medical treatment the prognosis is unfavourable. The stenosis of the bowel gradually increases until there is absolute obstruction or perforation into the abdominal cavity or adjacent organs. Death may be caused by pulmonary phthisis, peritoneal tuberculosis, by disseminated tuberculosis of the intestine, by the protracted suppuration of multiple abscesses, by amyloid changes in the intestines or kidneys, or by other complications.

Diagnosis and Differential Diagnosis

The detection and identification of ulcers of the intestine present many difficulties. One, already alluded to, is that many forms run a perfectly latent course or give but slight clinical symptoms. A second difficulty, which has also been mentioned, is that the different forms have no specific characteristic signs. With few exceptions, a positive diagnosis can only be made when the other clinical facts clearly show the relationship between and etiology of the symptoms. On the other hand, it occasionally happens that, although the standard symptoms are absent, one may suspect ulceration of the intestine when disorders of the functions of the bowels, pronounced tenderness, diarrhœa, and marked impairment of the general health are suddenly or gradually added to the previous symptoms. Even in such a case, however, the disease may only be suspected.

The recognition of ulcers of the small intestine, especially those which are not tuberculous, is especially difficult. The changes in the stools may perhaps consist in hemorrhage or melæna. The presence of pus can not be depended upon because, as von Leube[2] has shown, pus loses its characteristic appearance in passing through the colon. It is unnecessary to say that hemorrhages from the intestinal canal may have a great variety of causes. In a few cases, as in those reported by Nothnagel[23], the diagnosis of embolic and thrombotic ulcers has been successfully made, and is of course possible when the source of the embolus can be clearly traced. This may be possible when endocarditis, pyæmia, or arterial sclerosis has preceded, or when the symptoms of embolism of a branch of the mesenteric artery (severe colicky pains, severe hemorrhage setting in at once, meteorism, intestinal paralysis) have been present and, what is of course very rare, have abated.

The prospect of recognising ulcers of the large intestine (exclusive of the rectum) is better, because the excreted products are accessible to direct and repeated examination. Aside from the changes in the consistence of the stools and the local pain and tenderness (whose value must be estimated with care), the main factors to be looked for are admixtures of blood, pus, mucus, and tissue *débris* with the fæces. Naturally, the presence of both blood and pus is of the highest importance, while, as already explained, either of them alone leaves room for many possibilities. The diagnosis is not complete until not only the presence of an ulceration is determined, but its special causation made out. In a few instances, in addition to pulmonary tuberculosis, dysentery, and typhoid fever, there are no special difficulties in reaching a conclusion, but there are varieties which present insurmountable difficulties.

I shall relate one case in which the diagnosis of tuberculous ulceration of the large intestine was probable, and another in which, although ulcer of the intestine was diagnosed with positiveness, the etiology of the ulcer was never cleared up.

CASE I.—Miss Clara St., of Radenickel, near Crossen; twenty-eight years old.

Previous History.—Father had chronic pulmonary disease—otherwise no hereditary taint could be made out. The patient has been weak and sickly from her youth. Began to menstruate at fourteen; is regular, with very profuse flow. Six years ago, without any exciting cause, in particular without any previous cough, moderate hæmoptysis. This was repeated every four to six weeks. Two years later began to have pains in the epigastrium, which her physician attributed to gastric ulcer. She was sent to Carlsbad. Here she had hemorrhages from the stomach and melæna. In the following year hæmoptysis returned every four to six weeks. No change occurred until October, 1896, since which time there has been no hæmoptysis.

The present illness developed shortly before Christmas, 1897. It began with a sudden attack of severe pain in the region of the umbilicus, lasting day and night, not dependent upon the ingestion of food, and increased by pressure. Six weeks later there was a sudden discharge of pus from the rectum, preceding a normal passage. The pus coated the fæcal masses superficially. This discharge of pus was repeated four weeks later, the pains having meanwhile ceased. From that time on evacuations of pus continued at intervals of four to six weeks. During this period there were attacks of pain in the region of the intestines, but not simultaneously with the purulent discharge. These attacks used to last for a few weeks and then disappear for a few months.

Since May, 1898, the purulent discharge has been constant. There has been a normal painless stool every day. There are frequent cramplike pains, occurring only at night. By the end of May, bright fluid blood began to appear with the pus. This gradually increased in amount. Since the middle of June, 1898, the stools have been preceded by clear blood, and followed by blood with a little pus. During the last few days, neither pus nor blood has been passed.

Present Condition.—The patient is poorly nourished; the cheeks and lips are pale. Pulse, 72, of moderate tension. No fever. Nothing of especial interest in the lungs. The abdomen shows no abnormal resistance, no increased meteorism, no tenderness anywhere. Urine, negative; in particular, no indican. Examination by the vagina and rectum, negative.

Stools, solid and of normal calibre.

Irrigation of the bowel gave a negative result.

On June 30, 1898, she had a movement consisting of well-formed fæcal masses coated with muco-purulent shreds, in which pus cells were demonstrated in abundance by the microscope. Repeated examination failed to show tubercle bacilli. Digital and ocular examination of the rectum resulted negatively.

There is no doubt that ulcers were present in this case. It is most probable that they were located in the descending colon or the sigmoid flexure. In spite of the absence of signs of pulmonary lesions and the failure to find bacilli, the diagnosis of tuberculous ulceration of the large intestine is probable from the previous history of repeated hæmoptysis and the hereditary taint.

Case II.—Max B., clerk; age, twenty-eight years. Was perfectly healthy until 1895. In July of this year he acquired gonorrhœa, followed by stricture, which was treated with sounds and cured. In April, 1898, had another attack of gonorrhœa which lasted three weeks. During this period he states that he had "catarrh of the bladder." He had a chancre at the same time, which lasted fourteen days. Whether this was a soft chancre or an initial lesion, the patient does not know positively; but, at any rate, he was treated without mercurial inunctions by a specialist.

As early as January, 1897, the patient noticed for the first time bleeding from the bowel, the cause of which he does not know. There had been no antecedent constipation of notable degree. During this period he had twelve movements from the bowels each day, fluid, and mixed with blood. He does not know whether they contained any pus. There was no pain, either spontaneous or during defecation; and the actual loss of blood, in his opinion, was less than at the present time. Under the treatment which the patient received from January until July, the diarrhœa gradually ceased, the blood continued to appear from time to time, even when the stools were normal otherwise, and finally even this ceased. From that time until October, 1898, he had no abnormal symptoms. The treatment was by internal remedies.

The patient now appears, complaining that he has four to five diarrhœal passages per day which contain blood and mucus. He has tenesmus, and a fluid resembling bloody mucus frequently escapes without fæces. There is no pain. He has a good appetite, is easily satiated, but hunger returns in a short time. However, he does not feel in any wise ill.

Present Condition.—A moderately well-nourished young man of slender build, who looks healthy. An examination of the thoracic viscera shows them to be absolutely normal; there is no ground for suspecting pulmonary phthisis. The abdomen is nowhere distended, gives no abnormal percussion resonance, and is nowhere sensitive to pressure. Digital examination of the rectum gives quite normal results, confirmed by repeated visual examination. There are no signs of syphilis. There is no swelling of the glands, no leucoderma, no atrophy of the base of the tongue, and there are no nodules. (In response to an inquiry, the specialist who treated the case in 1898 says that it was undoubtedly a soft chancre.) The urine contains neither sugar, albumin, indican, nor peptones. The patient was directed to bring a sample of his passages at each visit.

The examination of the fæces, which was made on an average two or three times in each of the following weeks, showed that they were of normal colour, partly formed, but mainly pulpy. There was a slight superficial admixture of bright-red blood and a large admixture of greenish-yellow pus which settled in a thick layer at the bottom of the glass. There was no intimate mingling of the pus and blood with the fæces, and no further abnormal changes could be made out upon microscopic examination. *Repeated examinations for gonococci and tubercle bacilli were invariably negative.*

The treatment consisted at first in suitable diet, all irritating substances being excluded. Under irrigation with chamomile tea, and subsequently with a solution of tannic acid, the diarrhœa improved somewhat, but the blood and pus continued in variable quantity. There was no pain, either spontaneous or on pressure. Irrigations with a solution of nitrate of silver, 1 to 1,000, were tried, but had to be abandoned before long because they increased the diarrhœa, and the patient complained of burning sensations in the rectum and sigmoid flexure. Illumination of the rectum, the patient being in the lithotomy position, again failed to give any positive result.

December 10, 1898.—The blood and pus in the stools have increased. Patient says that on alternate mornings he has four or five movements of the bowels following close upon each other; they contain a considerable amount of pus; and that on the intervening days the conditions are nearly normal.

December 18th.—Enema of subnitrate of bismuth in suspension, preceded by a cleansing enemata of chamomile tea.

December 29th.—Has only one or two movements from the bowels daily, of thin, pasty consistence and still containing blood and pus in variable quantity, but always enough to be recognised by the naked eye.

January 17, 1899.—Patient feels very well. One or two movements from bowels daily. They still contain blood and pus, but in smaller quantity. No pain. Abdomen not tender on palpation at any point. Slight meteorism on percussion below the umbilicus.

In this case also, the diagnosis of ulceration of the large intestine is a safe one; but it is not possible to determine the nature of the process (whether chronic dysentery, follicular ulcer, or tuberculous ulcer). There could not have been a follicular or catarrhal ulceration at the bottom of this case, for the suppuration was too extensive; and there was besides an absence of a long-continued antecedent catarrh to act as a predisposing cause.

In the diagnosis of ileo-cæcal tuberculosis, the following points should be considered: The recognition of a tumour in the ileo-cæcal region, the youth or middle age of the patient, the presence of pulmonary or other localization of tuberculosis, a long duration of the symptoms, emaciation and pallor, the presence of tuberculous processes in other organs (lungs, joints, etc.), and, finally, the existence of an intestinal stenosis having the features first described by König[19]. They are the following: The abdomen is distended; frequently there is visible peristalsis, accompanied by gurgling, splashing, sometimes musical sounds, especially in the neighbourhood of the cæcum. Toward the end of an attack the sounds heard resemble those produced by expelling the last drops from a syringe; the abdomen collapses and the attack is over. According to König, these peculiar manifestations depend upon the relation between the length and the tightness of the stricture, the hypertrophy of the portion of the intestine above the constriction, and the relaxation of the gut below it. In such cases Ehrlich's diazo reaction gives important confirmatory evidence, for the extended investigations of Krokiewicz[24] have demonstrated that this reaction is almost invariably negative in carcinoma of the digestive tract, while it is seldom absent in tuberculosis. In addition, as Obrastzow[25] has shown, the finding of tubercle bacilli may fortify the diagnosis. Aside from carcinoma, a differential diagnosis must take into consideration the possibility of exudations in the ileo-cæcal region, especially perityphlitis, and also various rare forms of tumour (sarcomata, fibromata, foreign bodies, intussusception, fæcal tumours, actinomycosis, pericolitis, tumours consisting of abnormally located or displaced segments of the bowel, etc.). It is impossible to enter into a full discussion of all these sources of error, and these hints, although they by no means exhaust the possibilities, must suffice. The differential diagnosis between carcinoma and tuberculosis of the cæcum calls for special notice. Experience teaches us that it is very difficult. None of the above criteria, even the finding of tubercle bacilli, insures us against error. Only a collective consideration of the signs of cæcal tuberculosis warrants a decision. The following table will be of service, though, like all such schematic presentations, it has only a limited value :

Tuberculosis of the Cæcum	*Carcinoma of the Cæcum*
Age: Usually the second to fourth decades.	Seldom before the fourth decade.
Duration: Extremely chronic.	Duration that usual for carcinoma.
Lungs: Frequently more or less pronounced tuberculosis.	Examination of the lungs negative.
Tumour: Considerable extension in the length of the intestine; the infiltration can be shown by palpation to involve the bowel.	The tumour has a definite outline, which is usually strictly limited to that of the cæcum. The latter can not be felt as such.
Symptoms of stenosis: Always present; distinguished by remarkable murmurs.	Symptoms of stenosis may be entirely absent; when present, are usually less pronounced than in cæcal tuberculosis.
Condition of the stools: Blood and pus very seldom; tubercle bacilli very often present.	Blood and pus are not infrequently found; never any tubercle bacilli.
Fever: Not infrequently observed.	Fever exceptionally present.
Urine: Ehrlich's diazo reaction is present.	Diazo reaction always negative.

TREATMENT OF INTESTINAL ULCERS

In the treatment of intestinal ulcers, other than those of the rectum, the curative measures at our command are few in number. The weapons which asepsis and antisepsis have furnished so abundantly for the cure of external ulcers are useless in the treatment of intestinal ulceration. Since we lack an agent which has an efficient and lasting influence upon the intestinal juices and the intestinal contents, we can not even fulfil the simple postulate of *nihil nocere*. The only thing we can do is to avoid strong irritants, and restore the functions which have been perverted by the ulceration or its accompanying catarrh to normal.

This is especially true of *ulcers of the small intestine*, and among these of the tuberculous. Here our chief task lies in the control of the diarrhœa, partly by diet (see page 224) and partly by the astringent agents already described (page 191). We again call attention to the favourable action of the preparations of chalk, either alone or in combination with bismuth (dermatol, beta-naphthol-bismuth, etc.). In some cases of severe tuberculous diarrhœa in which the diagnosis of ulceration was made with as much positiveness as it can be at the present day, I have been able to keep the profuse evacuations at least temporarily under control by rest in bed and anti-diarrhœal diet. It is hardly necessary to state that this treatment is sometimes ineffectual.

In *ulceration of the large intestine* the results are more favourable, as we may supplement diet by direct local treatment with suitable irrigations. This measure, however, should not be valued too highly, since the agent is only briefly in contact with the diseased area, and the reaction of the bowel to various drugs is found by experience to be very much increased. Besides, in chronic diarrhœa it is not possible to keep the intestine in a clean condition. These are all factors which make the utility of local treatment, at least in severe cases, to some degree problematic. Nevertheless, the indication in every case is to try suitable disinfecting and astringent drugs: such are boric acid, 3 per cent; salicylic acid, 3 per cent; salicylate of soda, 5 per cent; nitrate of silver, 0.2 to 0.5 to 1 per cent; tannic acid, 0.5 to 1 per cent. In some cases I have seen decisive results from bismuth injections given in similar way to Fleiner's method in gastric ulcer. I use one teaspoonful to one quarter litre of water. On account of the absence of any irritating quality, I prize this drug above all others, and recommend that it be given the preference.

The treatment of *ileo-cæcal tuberculosis* is surgical. The results are temporary, of course, since no small number of the patients sooner or later fall victims to pulmonary tuberculosis. Of 86 cases reported by Conrath[14], the 23 operated on were found to be in good health one to four to eight years later. Judging from experience, the dangers of the operation are not inordinately high. According to Conrath, in 86 operations the mortality was only 16 per cent. The surgical procedures that have been tried are the extirpation of the tumour by resection of the intestinal wall, or by intestinal anastomosis (after Maisonneuve), or without extirpation the complete exclusion of the involved segment (division of the ends of the excluded portion of gut after the method of Salzer). Experience shows that entero-anastomosis offers the best chance of recovery (Conrath's statistics give 10 cases without a death), and the results otherwise are equally as good as after extirpation. In the future it should be the operation of preference.

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CHAPTER XVI

ROUND ULCER OF THE DUODENUM

(*Ulcus rotundum duodeni*)

Preliminary Remarks.—Among the ulcerative processes of the intestinal canal round ulcer of the duodenum demands careful attention—anatomically, because of its size and marked characteristics; clinically, because of its obscure symptomatology and diagnostic signs; and because of the severe complications which may mark its course and appear with an extremely acute onset.

In its most important features the pathological anatomy and pathogenesis of ulcer of the duodenum is the same as that of gastric ulcer. The extremely voluble discussion which followed Cruveilhier's classic presentation of the latter subject has not solved its numerous problems. With reference to the mode of origin of chronic duodenal ulcer we also are obliged to fall back upon more or less well-sustained hypotheses.

Referring the student of this subject to the text-books on diseases of the stomach, as well as to the monographs of Krauss[1], Chvostek[2], Boucquoy[3], Oppenheimer[4], Reckmann[5], and Collin[6], we shall, in what follows, limit ourselves to a concise description of the most important etiological factors which we have gathered from literature and from our own experience.

In the first place, just as in the case of gastric ulcer, so predisposing and immediate causes operate in the production of the duodenal ulcer. As regards the predisposing causes, it is universally, and I believe correctly, held that the corrosive action of the gastric acid, which is not neutralized to any essential degree until it meets with the pancreatic secretion, plays an important part. The fact that round ulcers of the small intestine occur almost exclusively in the duodenum permits scarcely any other explanation. But there are other predisposing causes. Dickinson[7], and shortly afterward Perry and Shaw[8], as well as Marmaduke Sheild[9], have called attention to the appearance of duodenal ulceration in the course of chronic nephritis. Thus, from the literature of the subject, Perry and Shaw have collected 70 cases of duodenal ulcer, in no less than 12 of which typical Bright's disease was present. In such cases everything points to a necrotizing effect exerted by the retained urea or its derivative, ammonium carbonate. It is apparent that in this case we have to deal with the same influences which produce multiple uræmic ulcers in the lower segments of the bowel. The toxic influence may act continuously or suddenly, perhaps after extirpation of one kidney, or after a severe acute nephritis with suppression of urine.

A different set of conditions underlie those forms of duodenal ulcer which have been observed after extensive burns, after frostbite, and in erysipelas, pemphigus, and septicæmia. The most likely explanation for the first-named condition seems to be that soluble fibrin ferment gains access to the circulation and emboli are formed, resulting in a partial necrosis of portions of the duodenal mucous membrane. On the other hand, in the case of the infective processes named, bacterial influences doubtless play an essential rôle. In still other cases a traumatic lesion may with more or less probability be looked upon as the original factor. Such cases have been observed by Schulze[10], Brambillo[11], Reckmann[5], J. Pauly[12], and others. Thus it may be seen that very varied causes may operate to produce the same anatomical changes, and it is of importance clinically to discriminate between them.

To these etiological memoranda may be added a few brief data on the age and sex affected, and on the localization of duodenal ulcers.

As far as *age* and *sex* are concerned, the duodenal ulcer shows striking variations from gastric ulcer. Collin, to whom we owe the most complete collection of cases (279), gives the following summary:

Under 10 years	42	cases.
From 11–20 years	24	"
" 21–30 "	43	"
" 31–40 "	52	"
" 41–50 "	46	"
" 51–60 "	41	"
" 61–80 "	18	"
" 81–94 "	13	"

Of the cases occurring in the first ten years nearly one half (17) belong to the first year of life. Duodenal ulcer has even been observed in newborn children who have only lived a few hours, so that an intra-uterine origin has been suspected. Landau[13] has ascribed them to thrombosis of the umbilical vein and embolism of the vessels of the small intestine, with consecutive necrosis.

The infrequency of duodenal ulcer at puberty, its slow increase during the third decade, its marked rise in the fourth and fifth, and its slow falling off again in the sixth, is noteworthy.

There is a striking unanimity among the various authors as regards the preponderance of the affection among the male sex. Collin found 205 cases out of 257, or 79 per cent, in males. This fact is one of the most striking in the pathology of duodenal ulcer. How shall we explain the fact that the male sex, which has so marked an immunity from round ulcer of the stomach, should show so peculiar a predisposition toward duodenal ulcer? In my opinion the explanation can only be found in the difference in the habits of men and of women. Of especial importance is the fact that the use of alcohol and tobacco more often causes a chronic gastritis with marked hyperacidity in men than in women. This, however, would not explain why the duodenum becomes the favourite seat of ulceration. In this connection the following considerations appear to me to be worthy of notice.

Through investigations on dogs made by von Mering[14] and Moritz[15], we know that the stomach expels water into the intestine with extraordinary promptness; and further, that alcohol, salines, dextrin, and acids, although they are absorbed by the stomach, are only taken up by it to a very limited extent. It further appears, from the investigations of von Mering, that the first discharges contain these substances in very concentrated solution. When von Mering poured 300 cubic centimetres of 25 per cent alcohol into the empty stomach, 105 cubic centimetres of 10.5 per cent alcohol flowed out within ten minutes. When he poured 200 cubic centimetres of 50 per cent grape sugar solution into the empty stomach, the fluid which was carried into the duodenum amounted to 120 cubic centimetres, with 32 per cent sugar. In addition, we know from Moritz's investigations that the stomach first expels the fluid portions of the chyme, while the solid portions follow quite slowly. Furthermore, the stomach protects itself from highly concentrated solutions by secretion of water; such a function has not yet been demonstrated for the duodenum. It thus follows that the duodenal mucous membrane has much less protection against concentrated watery solutions than the stomach, whose mucous membrane is relatively well protected against injury during the first stage of digestion by the slippery or solid contents. If, then, acids, alcohol, and saline solutions act upon a duodenal mucous membrane already irritated by an existing hyperacidity, it only needs an opportune cause to produce a partial necrosis of this unresisting tissue. This explanation receives a further illustration from the observations of Boucquoy[3], Burwinkel[16], and others, which I can confirm, that ulcer of the duodenum occurs with especial frequency in habitual alcoholics. If I might cite the results of treatment as a guide to an opinion in this matter, I would assert that duodenal ulcer, although not always in classic form, is a very prevalent lesion in alcoholics. I shall return to this point in the section devoted to symptomatology and diagnosis.

Among the features worthy of notice, we would call attention to the fact that duodenal ulcers, like gastric ulcers, are usually single. Out of 233 cases in which Collin found the number noted, the ulcer was solitary in 195 (83.6 per cent). Occasionally duodenal ulcer is found associated with gastric ulcer or with esophageal ulcer. In the great majority of cases the ulcer is situated in the upper part of the duodenum (242 times in Collin's table of 262 cases); in not a few (74) it was adjacent to or in contact with the pylorus; in 14 cases the seat was the descending portion, and in only 6 was it found in the inferior horizontal portion.

There is a discrepancy in the statistics as to the relative frequency of involvement of the anterior and the posterior wall. Oppenheimer states that it is as 18 : 16. Collin found that out of 127 cases in which particulars were given, the ulcer was on the anterior wall 71 times, on the posterior wall 45 times, on the upper edge 10 times, and only once on the lower edge ("*Bord superieur ou inferieur*"). In the descending portion the inner wall was most often involved, especially in the immediate vicinity of the papilla.

The duodenal ulcer not infrequently gives rise to numerous and serious complications, which will be comprehensively considered further on.

Symptomatology and Diagnosis

In the first place, it is to be noted that a duodenal ulcer frequently runs its course without any symptoms, and that it may cause death by perforation into the abdominal cavity at a time when the patient seems to be in perfect health. Such a latent course is apparently more common than it is with gastric ulcer. Whether in such cases there have not been slight symptoms extending backward over perhaps a long period is difficult to determine, but the fact remains that perforation is seldom preceded by severe gastric or intestinal symptoms. Such cases have been termed acute ulcers.

The symptoms may be divided into subjective and objective.

A. *Subjective Symptoms*

Pain.—The pain of duodenal ulcer closely resembles that of gastric ulcer. It is of a burning, boring character, and radiates downward or to the sides, seldom or (as Burwinkel says) never toward the back. According to Oppenheimer, the pain is increased by lying on the right side. A characteristic feature of the pain is that it comes on several hours after the ingestion of food, and is localized in the right hypochondrium at a point on the prolongation of the parasternal line about two centimetres below the gall bladder. There are many exceptions to this. The pain may be more to the left in the pit of the stomach, in the umbilical region, or exceptionally it may be more or less below this point. Judging from my own experience, I should also state that there is no relationship between the nature of the food and the onset of the pain, and also that the latter may persist, even begin during the fasting state—for example, at night.

From observations which he made, Chvostek formulated a test which might serve to differentiate duodenal from round gastric ulcers. He found that pressing gastric pains coming on two and a half hours after breakfast were permanently, and similar pains about three hours after dinner were temporarily relieved by the taking of wine. He concludes from this that when the ulcer is in the duodenum, the taking of the wine causes a reflex closure of the pylorus, and thus arrests the flow of the gastric contents into the duodenum. This, in the case of the more abundant meal, causes a temporary remission, and in the case of the lighter one a lasting relief. But if the ulcer is situated in the stomach, the swallowing of wine not only does not relieve the pain, but increases it. Similarly Burwinkel[16] reports a case in which "pain in the stomach," beginning two or three hours after each meal, was relieved by the taking of an acid wine or citric acid.

With certain limitations I have been able to confirm this sign in several cases of duodenal ulcer. I believe that it is dependent solely upon hyperacidity of the gastric juice, for we know that in this condition the ingestion of fluid or food will cause a cessation of the pain. Whether this comes about, as Chvostek thinks, from a reflex pyloric closure which for the time being prevents the passage of the food into the duodenum, or, as I believe, from dilution of the superabundant hydrochloric acid by the fluid and consequent diminished irritation of the ulcerated surface, may be left an open question. At any rate, it seems to me that the nature of the fluid is of no importance except that substances such as milk or egg albumin, which have a strong tendency to combine with hydrochloric acid, should act better than, for example, wine.

B. *Objective Symptoms*

1. *Points of Tenderness.*—The typical point of tenderness on pressure coincides with the area of spontaneous pain above described, but, like it, may exhibit numerous variations which may easily lead to errors in diagnosis. As far as I know, a dorsal point of tenderness has never been observed. In several cases I have noted a circumscribed tender point to the right of the spinal column and close to the twelfth dorsal vertebra.

2. *Vomiting.*—When the pain is severe and long continued it may lead to vomiting, which is probably of a reflex nature. Strange to say, there are only scanty allusions to this symptom in recent literature. Oppenheimer found it noted 17 times in the cases (over 100) which he collected.

The accounts given by older authors (Albers[17], Mayer[18]) do not agree with our experience of to-day. Krauss more correctly observes (loc. cit., p. 59): "In the clinical histories which I have collected it [vomiting] is very seldom mentioned; it depends either upon stricture of the duodenum or is the result of cardialgia. In a few cases only is it seen in connection with dyspeptic phenomena." Starke[19] and Boucquoy[3] make similar statements. My own experience, as far as it is possible to draw conclusions from a few observations, confirms their views.

The vomited matter has been variously described. In one observation reported by Reckmann[5], which in my opinion is open to some criticism, the vomiting occurred in three installments—the first pale and watery, the second bitter and sirupy (bile), and the third a sweetish mass (blood).*

3. *Intestinal Hemorrhage and Hæmatemesis.*—Profuse hemorrhages from the stomach or rectum are a frequent symptom of

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* See under head of the Examination of the Gastric Contents (in Special Part).

duodenal ulcer (perhaps in one third of all the cases—Krauss, Chvostek, Oppenheimer). It is probable that this figure is too low, as many of the smaller hemorrhages no doubt escape observation. In 34 cases of hemorrhage which Oppenheimer found recorded, vomiting of blood occurred 8 times, malæna 10 times, and both hæmatemesis and melæna 16 times. In all the severe cases symptoms of collapse follow the escape of blood by the mouth or the bowel. When very copious the hemorrhage may be the immediate cause of death. It is very characteristic of duodenal ulcer that the intestinal hemorrhages recur at fixed intervals coincident with other phenomena of the ulceration.

4. *The Composition of the Gastric Contents.*—There are only a few observations on record (von Leube [20], Reckmann [5], A. Robin [21], and Devic and Roux [22]). In the first two there was a condition of subacidity. In the last mentioned, a case accompanied by progressive pernicious anæmia and profuse diarrhœa, there was hyperchlorhydria. The latter condition was present in one of my cases, though it should be stated that the examination was made a long time before the occurrence of the intestinal hemorrhages. Until more observations have been accumulated, a differential diagnosis on the basis of the results of examination of the gastric contents is not permissible; the same opinion is expressed by von Leube. In three cases Robin found an entire absence of free hydrochloric acid, but an abundance of organic acids. Unfortunately no details are given as to the kind of test meal used, nor of the motor activity. Nevertheless Robin's results are very remarkable.

5. There is nothing characteristic in the urine or the dejections.

6. *Icterus.*—The jaundice which has been observed in a few cases (according to Collin, 9 out of 262) is not a specific sign of ulcer of the small intestine; it belongs rather to the complications (see below).

Of all these symptoms, not one *per se* enables the diagnosis to be made with certainty or even probability. Only the *ensemble*, the entire clinical history, the consideration of age and sex, is significant or decisive. Taking into consideration all of these symptoms (which are not often associated in a single case), and excluding all other possibilities, the diagnosis of an ulcer of the small intestine may be clinically made with some degree of certainty. In this view I agree with Chvostek, Boucquoy, and Burwinkel, but there are many authors who are of a different opinion (von Leube, Ewald, Eichhorst, Nothnagel, Collin). It is indeed pushing scepticism to the limit to say, as does Collin in his otherwise admirable thesis, that the diagnosis of duodenal ulcer *intra vitam* is impossible.

What I have just said will be illustrated by two positive, one probable, and one doubtful cases of duodenal ulcer.

1. Secretary of Police R., of Berlin, comes from a healthy family, in which hæmophilia has never been observed. No history of any previous illness. Took sick about twenty years ago (1876) with anæmia and tarry stools. Previous to that time there had been gastric and intestinal disorders. The patient subsequently recovered, and aside from temporary attacks of indigestion, was fairly well until 1891. At this time, after having suffered from a sense of oppression in the region of the stomach, he suddenly had several evacuations of very black stools. The patient fainted at the time. For several weeks he was under treatment at the Augusta Hospital. Following this there was a long period of good health, although from time to time there were attacks of pressure in the umbilical region or in the pit of the stomach, and several small hemorrhages, to which little notice was paid by the patient. Another severe hemorrhage occurred in 1896, also with syncope. This, like the other, was recovered from. In 1897, while under treatment at Carlsbad, he had a return of the bleeding, which, however, did not last very long. From this time the patient never fully recovered his health. He has almost constantly a feeling of pressure and weight in the stomach and abdomen, not dependent upon the ingestion of food, and suffers from frequent eructations which are sour, but never putrefactive. In November, 1897, he had another severe hemorrhage. During the summer of 1898 he took the Wildungen cure for a catarrh of the bladder. In October, 1898, had tarry stools to a moderate extent, and in December, 1898, a severe hemorrhage. These were always preceded by a sense of pressure, fulness in the abdomen, and eructations of gas.

The patient does not remember ever having vomited or having had *severe* pains in the stomach or the intestines. The appetite has generally been good, but diminished after the attacks.

Present condition (abstract): Very anæmic—general nutrition much depreciated. Organs of circulation and respiration normal. Heart sounds clear, no adventitious sounds. Abdomen markedly relaxed; the integument may be raised in folds. Under suitable illumination the stomach is appreciable; it is apparently displaced downward, giving marked splashing sounds as far as three fingers' breadth below the umbilicus. The other abdominal conditions are normal, and in particular there is no tenderness either in the region of the stomach or duodenum. Examination of the urine shows it to be normal.

2. Oscar S., dealer in wood, Berlin, thirty-seven years old. The patient's mother was a chronic sufferer from gastric disorders, and was extremely emaciated when she died. His father is healthy. Since he was nine years old the patient has suffered from digestive troubles. At the beginning he used to have occasional pains in the gastric region, coming on without apparent cause and lasting a quarter to half an hour. His appetite was good and the bowels regular. When he was almost thirty years old the pains increased, radiated toward the right side, especially toward the back and to the right shoulder blade, and became more frequent. They usually came on three to four hours after eating,

occasionally while fasting, and very often during the night. The sort of food ingested made no essential difference, for the pain appeared equally after either fluid or solid diet. Rest in the dorsal position alleviated the pains, while active exercise increased them. There was never any vomiting. Appetite and bowels were always in good condition.

Under suitable diet, rest, and the use of Carlsbad water, sometimes at the Springs and sometimes in Berlin, his condition gradually improved. In January, 1896, he again began to have severe pains of the character described above; they extended backward and came on several hours after eating. He always felt well immediately after eating. This attack was followed by a slow improvement. In January, 1896, while travelling, he had a sudden *profuse hemorrhage from the intestine.* The blood was at first diluted and mixed with fæces, but subsequently there was clear blood of coal-black colour. At the same time there was extreme prostration, so that the man, who was of herculean build, was obliged to take to his bed and remain there for thirty-six hours. He was treated by rest in bed, and poultices, and later drank Carlsbad Mühlbrunnen. Improvement was rapid, and for a year and a half he was perfectly well. In October, 1897, the pains returned in the right side several hours after eating, and were relieved by the ingestion of warm food. The treatment for ulcer, carried out for several weeks, was again followed by improvement. In the summer of 1898 he took the Carlsbad treatment with good results.

The examination of the gastric contents on two occasions showed marked hyperacidity (HCl 0.28-1.25 per cent). Besides this, there was a characteristic tender point in the prolongation of the right parasternal line. At the last examination, November, 1898, the duodenal region was absolutely free from tenderness.

3. Joseph L., bookkeeper, born in Poland, thirty-one years old. The family history has nothing of interest. At the age of sixteen the patient had typhoid fever, and at eighteen cholerine. Since he was nineteen years old he has had gastric symptoms, which consisted in pressure in the pit of the stomach, frequent eructations, and marked constipation. For the past seven or eight years he has had hemorrhages from the intestines, as he says, every spring and fall. These are preceded by sudden extreme weakness, nausea, perspiration, and a desire to defecate. The first movement is free from blood, but those which follow are intimately mixed with blood of a coal-black colour. Has never vomited blood. During the past year there were four such hemorrhages. In consequence of the frequent losses of blood the patient has become very anæmic, and has not recovered from them as he used to.

From the notes taken when he was seen for the first time, only the following need be quoted: Palpation of the abdomen shows that on the right side above the umbilicus, about two fingers' breadth below the region of the gall bladder, there is a decidedly tender point, while the corresponding area on the left side is absolutely painless. The patient identifies this point as the seat of his pain, which is pressing in character, but never colicky. Aside from poikilocytosis, no changes are noted in the examination of the blood.

It is perhaps worth recording that a few ova of the trichina spiralis were observed, but this probably has no bearing on the present illness.

4. Mr. V., member of the Board of Accounts of Gross Lichterfelde, near Ber-

lin, forty-one years old. Well until December, 1897. Since then, without any evident cause, he has had a sense of fulness a few hours after meals. An examination which I made at that time gave absolutely negative results. On January 6, 1898, he was suddenly taken with severe syncope and melæna. On January 7th the syncope was repeated. On January 8th, after drinking milk, there was a slight vomiting of blood. Rectal feeding was resorted to. There was no further hemorrhage. Gradual return to health. Spent three months partly in the sanitarium and partly in the mountains.

He still has occasional pains on the right side of the median line, usually a few hours after eating or after physical exertion. The painful area varies: it may be more to the right or toward the median line, and may even pass over to the left side. The pain is cut short by taking more food or alkalies. There are times when there is no pain. Appetite good, bowels confined. *Examination shows that there is no sensitive area over the stomach, or to the right or the left of it, nor is there any dorsal tender point.*

The symptoms of the first and second cases fulfil all the requirements for an exact diagnosis. The chronic course, the repeated attacks of hemorrhage per rectum, and the absence of any special dyspeptic symptoms, leave scarcely any doubt as to the nature of the trouble. The diagnosis of the other two cases is more difficult. In the third, the repeated attacks of melæna without any special gastric symptoms, and the sensitiveness on pressure to the right of the pylorus, make duodenal ulcer probable. Some of the other characteristic symptoms, such as occasional pain significantly localized, are absent, so that some doubt remains.

The fourth case can not be decided offhand with any certainty, because the painful area is not fixed, the objective signs are absent, and there is hæmatemesis as well as melæna. The nature of the pain, coming on several hours after eating, might equally well be ascribed to hyperacidity.

DIFFERENTIAL DIAGNOSIS

It will be seen from the above histories, and still more so from the autopsy records of cases of this class, that the differential diagnosis is often very difficult, especially when there is neither hæmatemesis nor melæna. In such cases many physicians prefer not to commit themselves to a diagnosis or to attempt a differential diagnosis. I think this is going too far. If we were to wait for the appearance of hemorrhage in gastric ulcer, at least 30 per cent of all the cases would remain undiagnosticated, and therefore uncured.

In my opinion the greatest difficulty lies in distinguishing ulcer of the duodenum from gastric hyperacidity. Very often both diseases give the same symptoms: pain several hours after eating, relieved by the ingestion of food or alkalies, and localized at the pylorus or in the duodenum. The pylorus and duodenal region may be more or less sensitive to pressure in hyperacidity. I think that when the latter cases do not improve upon a diet suitable to that condition, it would be well to begin treatment as for ulcer as soon as possible. I have done so several times, with the result that from that time on the patients were free from their discomforts. Just as in gastric ulcer, von Leube's treatment has a certain value for the differential diagnosis of doubtful cases, so, in long-established cases of hyperacidity with symptoms suggestive of ulcer of the duodenum I would recommend that the treatment for the latter condition should be instituted experimentally.

As in gastric ulcer, the differential diagnosis between duodenal ulcer and irregular cholelithiasis, with or without icterus or cholangitis, may be exceedingly difficult. Icterus as well as intestinal hemorrhage are not uncommon in cholelithiasis. In his Clinical Study of Cholelithiasis (p. 130 et seq.), to which we refer the reader for further information, Naunyn has described the various conditions in which the latter affection may be associated with intestinal hemorrhage. From a differential diagnostic standpoint the following must be noted: enlargement, tenderness, and tumefaction of the liver, the presence of a decided sensitiveness on pressure over the posterior surface of the liver (in the neighbourhood of the twelfth dorsal vertebra), and sometimes an intermittent pyrexia. In complicated cases it is difficult to avoid mistakes.

If the pains are atypical with frequent remissions and exacerbations, a correct diagnosis is merely a matter of accident. As an illustration of this I will give one of the numerous mistaken diagnoses which appear in clinical records—that published by Hadham[23]. A painter suffered from severe colicky pains with free vomiting, which, as his gums showed the lead line, were taken to indicate lead colic. Autopsy revealed an ulcer with sharp cut margins on the anterior wall of the duodenum. Two similar observations have recently been reported by Alvazzi-Delfrate[24].

When there are intestinal hemorrhages, the chances for a correct diagnosis are more favourable. The hemorrhage in itself is, however, not pathognomonic. The diagnosis can only be safely made when all the clinical symptoms are present, but even then it is often exceedingly difficult to distinguish duodenal from gastric ulcer. If we arrange the differential features in the order of their value, age and sex must be given first importance. Ulcer of the duodenum occurs with preponderating frequency during the third and fourth decades, ulcer of the stomach during the developmental age; ulcer of the duodenum is vastly more frequent in men than in women. In duodenal ulcer there is an absence of special gastric symptoms, such as anorexia and vomiting; ingestion of food does not increase the pain, but diminishes it if present; the pain is localized and does not radiate; hemorrhage per os when present at all, is scanty in comparison with the melæna; the hemorrhages are repeated extremely often, while in gastric ulcer they are not so common.

Although the treatment of both kinds of ulcers and the therapeutic results obtained are the same, an attempt should be made to distinguish between the two conditions, if possible, for, aside from the danger of perforation, the prognosis of duodenal ulcer is much more favourable than that of gastric ulcer, since the tendency to stenosis or carcinomatous formation (see p. 293) is markedly less.

Complications

Duodenal ulcer is noted for the remarkable number of its complications. Either because of their frequency or on account of the peculiar symptoms to which they give rise, some of these are of practical importance. The most important is:

1. *Perforative Peritonitis.*—In Collin's collection of 262 cases this was observed 181 times, or 69 per cent. The seat of the perforation—and this is of especial importance to the surgeon—like that of gastric ulcer, is usually on the anterior wall. The rupture may occur into the abdominal cavity and thus cause death by perforative peritonitis,* or else neighbouring organs—the liver, the pancreas, the gall bladder, or colon—may be encroached upon. In this way permanent adhesions or fistulous openings into the gall bladder or the colon are formed, or there may be an erosion of an important artery or vein and death from hemorrhage, or a subphrenic abscess with pyopneumothorax, may result.

Furthermore, a duodenal ulcer on the anterior or posterior wall may cause an abscess, and ultimately, by perforation of the abdominal wall, result in a duodenal fistula; or the ulcer may rupture into a cavity walled off by previous adhesions, which increase until the abscess is so encapsulated as to produce a plastic resisting mass. I have seen a case which I believe to have been of this sort.

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* As has been shown by a case reported by Bardeleben (Virchow's Archiv, vol. v, p. 2), the perforation need not always cause peritonitis. The fatal result may come about just as rapidly from shock, collapse, or hemorrhage.

All these possibilities are founded upon more or less numerous clinical observations described in the above-mentioned monographs on duodenal ulcer. Since it would take too long to describe each of them, the reader is referred to these authors for details. There are no specific symptoms which indicate the site of the perforation. The answer is most decidedly in the negative. There are accounts of operations (Bryant[25], Brissaud[26], Sheild[9], Lockwood[27], Lennander[28]) undertaken for supposed perforating appendicitis, in which the autopsies showed that the cause lay in a duodenal ulcer that had perforated. The error was usually due to the fact that the chief painful point was located in the ileo-cæcal region. It is hardly necessary to call special attention to the circumstance that a diagnosis of perforation is open to all the mistakes that may be made in the diagnosis of perforation of an ulcer in any other part of the intestine (strangulation, etc.).

2. *Icterus.*—Icterus is occasionally observed as a complication of duodenal ulcer, although Collin could collect only 9 cases in which it occurred. It begins as a true duodenitis, which involves the papilla of Vater. Cases of this kind have been seen by so competent an observer as Henoch, so that there is no good reason for doubting them. The jaundice has the well-known character of the catarrhal form, except that it is transient and does not cause enlargement of the liver. In a case reported by Krauss, the cause of the icterus was inflammatory adhesion of the duodenum to the gall bladder. Another form, more easily accounted for by what will shortly be described, occurs when the ulcer is located in the descending portion of the duodenum.

On account of its infrequency, icterus is of scarcely any importance for diagnostic or differential diagnostic purposes. Except under some favourable circumstances, it rarely throws any light upon the clinical picture.

3. *Formation of Stenoses by Cicatrization of the Ulcer.*—In a small number of cases the cicatrization of a duodenal ulcer leads to stenosis and consecutive dilatation of the parts lying above it—that is, of the stomach, or the corresponding portion of the duodenum. As the ulcer is most frequently in that part of the duodenum adjacent to the stomach, it is in the latter organ that ectasia is most often met with; according to Collin, 18 times in 262 cases. Duo-

denal dilatation was observed only 4 times. The symptoms of this condition will be considered in the chapter on Intestinal Stenosis. Lastly, in rare cases, when the ulcer is located in the neighbourhood of the ampulla of Vater, obliteration of the common bile duct and permanent jaundice may follow. This complication will make the diagnosis and the treatment very difficult.

4. *Carcinomatous Ulcer of the Duodenum.*—Carcinomatous degeneration of the ulcer seems to be rare. Altogether, but 4 cases have been observed (Eichhorst[28], Ewald[29], Mackenzie[30], Schrötter[31]). Unless it be that many cases are overlooked, this would constitute a striking variance from the frequency of carcinomatous ulcer of the stomach.

TREATMENT

The treatment of ulcer of the duodenum differs only in a few points from that of ulcer of the stomach. For the details of the latter I would refer to my Diagnosis and Treatment of Diseases of the Stomach, Part II, third edition, page 55, and will limit what follows to a brief sketch of the plan of treatment.

If hemorrhage set in, absolute rest in bed is the first requirement. The stomach should be put at rest, and for several days alimentation carried on by the rectum. Following this, the most promising course is von Leube's rest-cure treatment for ten to fourteen days, with the application of warm poultices, and a milk diet. After the cessation of the pain the diet may be cautiously increased week by week. Alkalies or Carlsbad water, bismuth or nitrate of silver, may be useful as adjuvants. In very obstinate cases it is advisable to give the stomach a rest by exclusive rectal feeding, under careful supervision, for a week or a fortnight. Opiates can not be dispensed with when the pain is severe. Long-continued physical rest, careful diet, and the avoidance of alcohol and tobacco are to be insisted upon.

When a cure is not obtained by palliative measures, and life is threatened by continuous pain or profuse hemorrhages, when there are symptoms of cicatricial stenoses not relieved by lavage, or when there is perforative peritonitis or subphrenic abscess, surgical procedures may be indicated. Owing to the analogy which these indications bear to those that arise in ulcer of the stomach, we refer the reader to the latest work of Mikulicz[32].

Up to the present time experience in the surgery of duodenal ulcer has been scanty. In one case Codivilla[33] excised the ulcer by a gastro-enterotomy with good result. Lange[34] performed a plastic operation on the pylorus in a case of cicatricial stenosis from duodenal ulcer, with equally good result.

Operations for perforative peritonitis have been successfully performed in several quarters (Herczl[35], Landerer and Glücksmann[36], Wannach[37]). In three cases reported by Lennander[38] a fatal termination occurred in spite of the operation.

In view of the absolutely hopeless prognosis of ulcer of the duodenum after perforation, surgical intervention should be undertaken as soon as possible—within ten to twelve hours at least after the diagnosis is assured and the primary shock has been recovered from.

Subphrenic pyopneumothorax or other abscess formations, are treated according to the prevailing surgical methods.

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CHAPTER XVII

INTESTINAL NEOPLASMS

A. MALIGNANT NEOPLASMS OF THE INTESTINES

I. Carcinoma

Preliminary Remarks.—Malignant tumours of the intestines are so very frequently carcinomatous that, in discussions relating to malignant new growths, cancer is almost exclusively the tumour in question. The other malignant tumours (sarcoma, lymphosarcoma), however, will require brief consideration because of the well-marked clinical pictures they occasionally present.

Regarding the absolute and relative frequency of intestinal carcinoma, there are many extensive and instructive statistics.

A. Zemann[1] found, in 21,624 autopsies performed at the Vienna General Hospital, 2,070 neoplasms directly causing death, of which number 1,744 were cancers—i. e., 84 per cent of all neoplasms.

Of these 1,744 cancer cases, 912 (52 per cent) were tumours of the gastro-intestinal canal, the "canal" in this sense beginning at the tongue and including the anal orifice.

The various portions of the gastro-intestinal tract were affected in the following proportion:

Tongue	37	
Pharynx	34	
Œsophagus	136	
Stomach	**540** *	
Duodenum	3	**165**
Ileum	6	
Cæcum	12	
Vermiform appendix	1	
Colon	32	
Sigmoid flexure	30	
Rectum	81	

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* I. e., 2.5 per cent of all the autopsies.

In his excellent work on the intestines, Nothnagel tabulates like statistics derived from a study of similar material. George Heimann[2] has recently published extensive tables which are very useful in the study of the pathology of the alimentary canal. During the years 1895 and 1896, 20,054 patients died of cancer in the general hospitals of Prussia, of whom 10,537 were cases of cancer of the gastro-intestinal tract. The different portions of the canal were affected as follows:

Tongue	269
Pharyngeal and buccal mucous membrane	192
Œsophagus	1,011
Stomach	**4,288**
Intestinal canal *in toto*	**1,706**
Of these, the rectum	**1,204**
Liver and gall bladder	979
Pancreas	92

Of the cases of cancer of the intestines (exclusive of the rectum), 20 involved the small intestine and 224 the large intestine; 49 of the latter affected the sigmoid flexure; in 258 cases the portion affected was not mentioned.

These statistics (which in the main agree with others) demonstrate the relative infrequency of intestinal cancer as compared with cancer of the stomach. The striking disparity in numbers between cancer of the small and that of the large intestine, and the overwhelming frequency of rectal cancer, are noteworthy. Finally, these statistics prove the frequency of cancer of the sigmoid flexure. Next in frequency to these are the carcinomata of the colon (especially at the flexures), and last of all those of the cæcum.

Regarding *sex*, authorities differ. Some believe that females are oftener affected (Bérard[3], Rokitansky[3], P. Rüpp[4]) while others regard intestinal cancer as more frequent in males (Maydl[5], G. Heimann[2]). It is certain, however, that cancer of the rectum occurs more frequently in men.

The age of the patient is an important consideration. Most cases occur between the fourth and sixth decades. As Maydl[5], Nothnagel, and G. Heimann[2] correctly pointed out, intestinal cancer quite frequently occurs in even the earlier periods of life; particularly is this true of cancer of the rectum, many cases of which have been observed in young people. In partial contrast to the above, sarcoma and lymphosarcoma occur usually during the first to the fourth decades, and only very infrequently in the fifth and sixth. Regarding *frequency*, sarcoma, in contrast to carcinoma, is oftener situated in the small intestine than in the large. (Compare chapter on Sarcoma.)

It will hardly be necessary to discuss at length the etiology of cancer and sarcoma, since, with the exception of the bacterial and protozoan theories, nothing of importance has been brought to light. On the other hand, histological investigations by modern investigators, particularly Virchow, Thiersch, Waldeyer, Hauser,* Ribbert, Hansemann, Lubarsch, and others, have proved more fruitful. A full description of these as yet incomplete investigations would lead too far, hence we will mention only a few pertinent facts. In the great majority of cases intestinal cancer is a primary affection, very rarely metastatic. The most frequent form is *cylindrical epithelial.* It originates, as do all other forms of intestinal cancer, from the cylindrical epithelial cells, and shows most often a glandular type (hence "carcinoma adenomatosum cylindro-epitheliale," Hauser, loc. cit.). The second most frequent form is the medullary (carcinoma medullare), which shows a decided tendency to break down and form ulcerations (intestinal hemorrhages). Somewhat less frequent (most often in the rectum) is colloid carcinoma. In striking contrast to the stomach, true *scirrhous* cancer occurs very rarely in the intestine, in most cases in the rectum.

The histological construction of the different types is not without its clinical significance. The soft, medullary type of cancer generally forms a diffuse tumour, while the scirrhous is a different growth, infiltrating the intestinal walls and being occasionally quite circumscribed. There are, however, exceptions to both these forms.

In its early stages cancer of the large intestine produces but slight changes in the mucous membrane ; only later does the growth show its special characteristics—propagation and tendency to ulceration. Another characteristic is its growth in a circular, girdlelike fashion. Isolated nodules (at times of large size), or long, diffuse infiltration, rarely occur.

From the two above-mentioned characteristics (viz., tendency to ulceration and circular growth of the tumour) we have the following two clinical conditions : A tendency to hemorrhage to purulent disintegration, even to separation of portions of the tumour and the development of stenoses. These symptoms will later be discussed at length.

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* Compare Hauser's interesting and critical description of the different theories of cancer in Das Cylinderepithel Carcinom des Magens und Dickdarms, Jena, 1890, p. 109.

Because of this tendency to ulcerate, superficial layers of the bowel may be involved in this destructive process, and perforation may occur. The development of a stenosis, which may at times cause complete obstruction, produces dilatation and muscular hypertrophy of that portion of the intestine above the stricture. Through overaction in this dilated and hypertrophied portion normal propulsion of contents is maintained, until a disproportion between the power of the hypertrophied wall and the resistance offered occurs, and an intestinal paralysis (ileus paralyticus), or, from extreme overexertion of the hypertrophied part, a rupture of the intestine at its weakest point results.

The peculiar characteristic of cancer to form metastases by way of the blood and lymph channels is also present in intestinal carcinoma.

The mesentery is supplied with lymphatics which lead to the mesenteric glands, and hence the latter are the first to become diseased, further metastatic involvement depending upon the site of the tumour. Thus, according to Rüpp[3], the lumbar glands are involved in cancer of the sigmoid flexure of the colon; the omental, and later the prevertebral glands, in cancer of the transverse colon.

Metastatic carcinoma of the lymph nodes may cause secondary intestinal obstruction, a condition which I have twice observed in cancer of the uterus. Disseminated carcinosis of the peritoneum is relatively frequent.

The observations of Maydl[5] and Hausmann[7], confirmed by Rüpp[3], are of extreme surgical interest. They assert that metastases are observed only late in intestinal carcinoma; this is particularly true of cancer of the rectum. Iverson[8] cites 47 autopsies of rectal carcinoma, of which 21 were free from secondary involvement of glandular organs; Kraske[9], in 12 cases, found metastases in only 6.

According to Hauser (loc. cit.), there is a connection between the type of carcinoma and location and variety of the metastatic formations. Thus colloid carcinoma very rarely causes secondary involvement of internal organs (e. g., the liver), but frequently secondarily involves the serosa, lymph glands, and bones. Large medullary carcinoma produces metastases principally in the regional lymph glands, while the small scirrhus may cause a large metastatic deposit in the liver. Besides the lymph channels, cancer may also spread by way of the blood-vessels. Tumour masses may erode the vascular wall (particularly of the veins), and thus infectious material enters the blood stream. Since its tributaries are directly in the intestines and are often connected with the malignant growth, the portal vein is especially prone to carry infection. For this reason the liver is often secondarily involved, while metastatic deposits in the lungs, uterus, and ovaries are rarer.

The tumour frequently becomes adherent to the neighbouring organs. Such adhesions may lead to complications, but they are favourable in so far as they prevent perforation. The most frequent complications of this kind are adhesions to and fistulous communication with the stomach, bladder, other portions of the intestines, the abdominal wall, genitals, etc.

General Symptomatology and Diagnosis of Intestinal Carcinoma

The symptoms of intestinal cancer vary so much according to its location that it seems judicious to consider separately cancer of the small intestines, the colon, and the rectum. A description of sarcomatous and lymphosarcomatous diseases of the intestines will be found in another chapter.

All cancers have certain general characteristics, which, although of no special significance individually, collectively complete the clinical picture of the disease. These are the history, the deportment of the body weight, the condition of the urine and blood, the occurrence of œdema of the ankles or of ascites, and, finally, the presence of infiltrated lymph glands.

1. *Hereditary disposition* to carcinoma is perhaps of value. Many striking examples of such a tendency have been described (e. g., family of Napoleon I). Traumatisms received during recent years should be noted in the history.

2. In obscure or atypical cases of intestinal cancer the *body weight* is of great importance, but is frequently not sufficiently considered. My experience has taught me that patients weigh themselves only when there has already been quite an apparent loss of weight, perhaps even as much as 10 to 20 pounds. At this stage, too, there is beginning cachexia. By weighing the patient at regular intervals the physician may often note the degenerative character of the disease much earlier. If the appetite be good and the condition of the upper portion of the alimentary

canal normal, forced feeding may be tried; if the patient then lose in weight there is probably some obscure malignant disease of the intestine, usually a neoplasm present. The progressive loss in weight should neither be over- nor underestimated, but regarded as a warning to examine the patient with greater care and frequency in order to recognise the obscure condition as early as possible. It must be remembered, however, that *increase* in weight does not by any means exclude carcinoma.

3. The *examination of the urine*, particularly the finding of indican and of Rosenbach's colouring matter may aid in making a diagnosis. The diagnostic significance of these bodies has already been dwelt upon (see page 132). Kast and Baas[10] attribute special value to the presence of ethereal sulphuric acids in the urine as a sign of intestinal putrefaction. This has occasionally been important to surgeons in determining whether a stenotic obstruction was successfully removed by operation or not. Rommeläre[11] and others drew attention to a lessened excretion of nitrogen by the urine in carcinoma, but the investigations of F. Müller[12] and G. Klemperer[13] have shown this conclusion to be deceptive.

4. Examination of the *blood* in cancer is of little diagnostic value. There is a diminution in percentage of hæmoglobin, accompanied by a corresponding decrease in the number of red blood-cells, and of the specific gravity of the blood. "Digestion leucocytosis," recently claimed by Schneyer[14] as diagnostic for cancer of the stomach, has been disproved by later investigations. As yet we have no reports regarding its occurrence in intestinal cancer.

F. Henry[15] has lately called attention to the differentiation between cancer of the stomach and pernicious anæmia by means of a count of the blood-cells. He has pointed out that in cancer the number of erythrocytes is never less than 1,500,000 per cubic millimetre, while in pernicious anæmia it is almost invariably below 1,000,000.

5. The early occurrence and disappearance of *œdema of the ankles* might, with a certain amount of reserve, be accepted as a contributory sign to the diagnosis of a malignant disease. I can not recall ever having seen it in any of my cases of intestinal cancer, and can find no mention of it elsewhere in the literature of the subject (cf. Intestinal Sarcoma, p. 332). Ascites is, of course, one of the most frequent accompanying symptoms of advanced cancer of the intestines. Once, during an operation for cancer of the cæcum, I observed slight ascites, which, because of its small quantity, it had been impossible to diagnosticate before operation.

6. Markedly enlarged inguinal or supraclavicular glands may lend support to the diagnosis. Swelling of the latter group is very rare, while enlargement of the inguinal glands, though frequent, occurs under most varied conditions. At all events, excision and histological examination may aid the diagnosis.

(a) CANCER OF THE SMALL INTESTINE

For the clinical diagnosis of cancer of the small intestine it is best to distinguish between cancer of the duodenum and that of the jejunum and ileum.

1. *Cancer of the Duodenum*

According to their relation to the papilla of Vater, these tumours of the duodenum are conveniently subdivided into suprapapillary, infrapapillary, and circumpapillary.

(a) SUPRAPAPILLARY CANCER

The symptoms of suprapapillary cancer are not well defined, and it is exceptional to arrive at even a probable diagnosis. Of some value are the *subjective symptoms* produced by the marked disturbance of gastric motility: the feeling of the tension and pressure or pain in the epigastric region, eructations, nausea and vomiting, loss of appetite, constipation, oliguria, marked thirst, and a decided feeling of illness and increased debility, sometimes sufficient to confine the patient to bed.

Of the *objective symptoms*, the earliest is the *presence of a tumour*. This lies in the right hypochondrium, is hard and uneven, painful on pressure, and is entirely immovable or only slightly movable.

The second objective symptom is the presence of a supraduodenal dilatation, which naturally leads to enlargement of the stomach. Here it will not be sufficient to diagnosticate simply anatomical dilatation of the stomach, but by repeated examinations of the stomach contents we must seek for proof of stagnation. As a recently published case of Czygan[16] has shown, the examination of the stomach contents for the presence or absence of hydrochloric acid, for lactic and other organic acids, for sarcinæ and yeast, lactic-acid bacilli, ferments, etc., may be of aid to the diagnosis. In the above-mentioned case, besides a tumour in the right hypochondrium and all other symptoms of cancer, there was found (except a few days before death) normal hydrochloric-acid secretion in the dilated stomach. I found the same chemical conditions in a case which I had under observation, and which, because of the absence of tumour, was diagnosticated as a benign pyloric stenosis. Laparotomy disclosed a tumour of the first portion of the duodenum as the cause of the stenosis.

We must, however, not lay too much stress upon the results of chemical examinations, since hydrochloric acid is often present for a long time in carcinoma of the pylorus, and, owing to the stagnation which results from proximity to the stomach, may be absent and lactic present in duodenal cancer.

Czygan[16] mentions another point of possible diagnostic value: the *presence of splashing sounds, particularly between the tumour and the free border of the ribs.* Upon emptying the stomach these sounds disappeared, but, upon filling the organ, were again observed, especially in the region above mentioned.

If an immovable or slightly movable tumour be felt in the right hypochondrium, by careful consideration of the above criteria we may venture upon the probable diagnosis of duodenal cancer, or, at any rate, arrive at the differential diagnosis between this disease and cancer of the pylorus. When no tumour is palpable, one can diagnosticate the gastrectasis, and, under favourable circumstances, the pyloric stenosis also.

(β) INFRAPAPILLARY CARCINOMA

Subjectively, infrapapillary carcinoma differs but little from the above-described suprapapillary type, excepting in one very marked symptom, viz., the vomiting of bile. When this occurs constantly it should arouse suspicion of an infrapapillary stenosis.

Objectively.—We again have an immovable tumour situated in the right hypochondrium, more or less distant from the free margin of the ribs, and painful on pressure. In addition there is the bilious vomiting, from which Leichtenstern[17] was the first to establish the diagnosis of carcinoma of the descending portion of the duodenum.* Examination of the gastric contents will frequently show the permanent presence of bile even before vomiting occurs.

As a result of the stagnation of contents, dilatation of the stomach may gradually be established, but because of frequent vomiting, mild stenosis, and early lavage of the stomach may be overlooked.

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* According to A. Pic, Chomel is said to have recognised and interpreted, as early as 1852, continued bilious vomiting.

At present it is impossible to state whether the examination of the gastric contents is of diagnostic or only contributory value. In an exhaustive study of a case, Herz[18] constantly found lactic acid present and hydrochloric absent in the strongly bilious stomach contents. The diagnosis of carcinoma of the stomach was made, but the constant presence of biliary matter might have attracted attention to the real seat of the lesion. Moreover, a tumour was not palpable. The presence of the tumour will not always verify a probable diagnosis. Thus, for example, Gerhardi[19], Hagenbach[20], Wilms[21], and others have shown that it is absolutely impossible at times to differentiate between infrapapillary carcinoma and tumours of the head of the pancreas. Diseases of the gall bladder—e. g., adhesions to the duodenum, compression by retroperitoneal glandular tumours, cicatricial strands following peritonitis, etc.—are conditions too complicated for differentiation.*

In isolated cases the history and clinical course of the disease are of some assistance. Previous melæna or hæmatemesis, signs of cholelithiasis or of an old duodenal ulcer, glycosuria, or fatty stools, may favour the one or the other diagnosis. Weecke[22] has pointed out that irritations or ulcerations of the walls of the common bile duct by gallstones may produce duodenal cancer, particularly the form next considered.

In the absence of tumour the diagnosis becomes quite uncertain. In such cases the age of the patient, emaciation, œdemæ, or ascites may point to the correct diagnosis, although the absence of severe general symptoms and the slow development of the disease may lead to a more optimistic conclusion. Such favourable conditions are, however, rare.

γ. Circumpapillary Carcinoma

In this the neoplasm develops in the neighbourhood of the papilla of Vater and compresses the bile duct. Whether in these cases we have to do with primary carcinoma of the papilla, or, as Pic[23] contends, with cancer of the pancreas, is certainly of scientific interest; from a clinical (particularly a diagnostic) standpoint this distinction is almost valueless. As discussion on this point is foreign to the subject at issue, it will not be further considered, and we shall at once proceed to the symptomatology of papillary carcinoma.

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* A case from Kussmaul's clinic, described by Cahn in 1886, was diagnosticated as duodenal cancer on account of the presence of a palpable tumour and continuous biliary vomiting. The autopsy showed a retroperitoneal lymphosarcoma compressing the descending portion of the duodenum.

Subjective Symptoms.—These are of secondary importance, viz., pain in the region of the stomach or liver, noncharacteristic but not biliary vomiting, constipation or diarrhœa, loss of appetite, and progressive loss of strength. One or all of these symptoms may be present, but they are of less value than the objective symptoms.

Objective Symptoms.—The main and most apparent symptom is *icterus.* Bard and Pic[24] have shown, however, that icterus may be entirely absent, and maintain that when a tumour and icterus are present the carcinoma generally has its origin in the pancreas. On the other hand, Lannois and Courmont[25] have published several cases of undoubted ampullary cancer with icterus. I myself have made two similar observations. According to my experience, the manner of development of the icterus is extremely important. As in catarrhal icterus, this is sudden, and usually without pain, but as Janicke has shown, severe attacks similar to gallstone colic may exceptionally occur, obscuring the true clinical picture. The difficulty of diagnosis is also illustrated by a case which I observed of cholelithiasis with a large gallstone in the common bile duct. The stone was passed without any pain, and the whole condition was accompanied only by chronic icterus. Unless sudden intercurrent complications—e. g., marked cachexia, hæmatemesis, melæna, and irregular fever—indicate the correct diagnosis, it will be impossible, therefore, in the first few days or weeks of the disease, to make a differential diagnosis between simple icterus, cholelithiasis, and beginning carcinoma

The longer icterus continues, the more resistant is it to the usual methods of treatment, and the sooner the general condition of the patient suffers, the more probable will the diagnosis of a malignant neoplasm become, particularly when the above-mentioned symptoms occur in middle-aged patients. In two cases of absolute anorexia under my observation, the total absence of response to customary therapeutic measures aided the diagnosis, because such extreme obstinacy is not found in either duodenal icterus or cholelithiasis. We also meet with this extreme anorexia in hypertrophic cirrhosis of the liver (which is otherwise sufficiently distinguished from circumpapillary carcinoma), and especially in pancreatic carcinoma, soon to be described. To my knowledge, no examination of the stomach contents in cases of circumpapillary carcinoma have been published. In one of my cases, despite absolute anorexia, there was no disturbance in the chemical or the motor functions of the stomach.

In circumpapillary carcinoma the liver is usually not enlarged, and the gall bladder is decreased in size. A tumour is rarely palpable, but when present it is too deeply situated to be distinctly outlined. Naturally, as in the case described by Kernig[27], the presence of a tumour is of great aid to the diagnosis; it may, however, be misleading, for it may simulate primary cancer of the gall bladder or liver. If icterus be absent, even in the presence of a tumour the diagnosis will be extremely difficult. It may be very difficult to differentiate between carcinoma of the papilla and of the pancreas. Bard and Pic[24] have recently published a set of symptoms which, recognised sufficiently early, is said to render the diagnosis of cancer of the pancreas as easy as that of cancer of the stomach. The symptoms are well-marked, constantly increasing icterus, with enormous dilatation of the gall bladder, rapid emaciation and cachexia, usually accompanied by subnormal temperature and absence of appreciable enlargement of the liver. For excellent reasons, in which I fully concur, Oser[28] has belittled the significance of this clinical syndrome.

Only the presence of sugar in the urine (according to Mirallié[29] this occurs in 26 per cent of all carcinomata of the pancreas) or of certain intestinal symptoms (bloody evacuations, hæmatemesis, diarrhœas) may point to the one or other condition in question. None of these symptoms are absolutely diagnostic.

2. *Carcinoma of the Jejunum and Ileum*

On account of its rarity and because, as stated by Treves[30], it very seldom forms a palpable tumour, carcinoma of the jejunum and ileum possesses but little diagnostic interest. Unlike cancer of the duodenum, tumours of this portion of the intestine are extremely movable. This characteristic may make it difficult to differentiate them from cancer of the large intestine, the difficulty naturally becoming greater the nearer the tumour is to the colon. As soon as stenotic symptoms appear, however, jejunal carcinoma may be diagnosticated. Enterorrhagia, when present, may be of value in making the diagnosis.

The subjective symptoms are very similar to those of duodenal carcinoma: colicky pains, increasing marasmus, anorexia, nausea, vomiting, and constipation alone or alternating with diarrhœa.

(b) CARCINOMA OF THE LARGE INTESTINE (EXCLUSIVE OF THE RECTUM)

Although cancer of the small intestine, on account of its infrequency, possesses comparatively little clinical importance the case is entirely different with cancer of the large intestine. In the latter, not only is there a question of diagnosis per se, but also of the earliest possible diagnosis, so that a radical surgical operation may be performed. For such early diagnosis it is necessary to have in mind not only classical cases, but also atypical, irregular forms of the disease.

1. *Typical Cases of Carcinoma of the Large Intestine*

Symptomatology and Diagnosis

Subjective Symptoms.—The most important are intestinal pain, vomiting, and disturbances of intestinal function. In concurrence with Rüpp, we distinguish the fixed or tumour pain from colic or other similar paroxysmal pains. In general, tumour pains are dull and localized; in some instances, however, they may radiate toward the back or the sides, toward the thorax, and, if the tumour be deeply situated, toward the legs. When not palpable, the site of the tumour cannot be determined by the localization of the pains.

The *paroxysmal pains* are of greater importance. Even in typical cases their situation, nature, intensity, and duration are extremely variable. According to my experience, the situation of the pain often points to the site of the tumour; occasionally, however, the pain radiates toward the umbilicus or over the whole abdomen. The pain is colicky in character, with remissions and exacerbations; it is slight at its beginning, becomes exceedingly severe as it reaches its acme, but soon decreases, to reappear after a longer or shorter interval. In one of my cases the remissions lasted precisely ten minutes, so that the patient, watch in hand, could exactly foretell the next attack. In other cases the paroxysms continue much longer, sometimes lasting for hours, with short remissions induced by eructations, passing of flatus, or by vomiting. In patients with complete stricture the pain is constant. Symptoms of ileus then develop. Rüpp[4] states that the pain is increased directly before defecation, especially in stenosis of the lower colon and when purges are given.

The picture is different in cancer of the lower bowel—i. e., from the lower portion of the descending colon downward. *Tenesmus* is prominent and characteristic. Since this constitutes one of the main symptoms of rectal cancer, further discussion will be deferred until the chapter on Carcinoma of the Rectum.

Constipation and *vomiting* are very intimately related to the paroxysms of pain. Constipation stands in direct relation to the pain: the severer the pain the longer the duration and the more obstinate the constipation. After a satisfactory evacuation the pain disappears; when the colon is refilled with fæces, paroxysms recur. If, in spite of fæcal movements, pain persists, we may be positive that the evacuation was insufficient.

In the first stage of the disease we sometimes find diarrhœa alternating with constipation, so that diarrhœa follows after several days' constipation. In still other cases constipation is entirely absent; from the very beginning diarrhœa of a nature later to be described is present.

The clinical picture of disturbed intestinal function is completed by the appearance of vomiting. The vomiting varies according to the degree of stenosis. It may consist of mucus, stomach contents, or of feculent or fæcal masses. The different types of vomiting demonstrate the amount of obstruction. With Rüpp, I think feculent vomiting, even in severe intestinal stenosis, is the exception. I am inclined to believe that the vomiting of mucus and of stomach contents is reflex in character, similar to that associated with impacted stone, cardialgias, disease of the genitals, etc.

In one of my cases of increasing symptoms of stenosis, hæmatemesis occurred directly before the operation. The patient fortunately withstood the shock of the hemorrhage. This is an extremely rare complication.

Leaving the last-mentioned symptom (hæmatemesis) out of consideration, there are three cardinal diagnostic symptoms, viz., obstinate constipation or constipation followed by diarrhœa, colicky paroxysms, and vomiting. Increasing experience has shown me that, even without the presence of a palpable tumour, these three symptoms frequently denote intestinal cancer. The frequency of the attacks, the characteristic increase of the paroxysmal pain, increasing constipation, the presence of unmistakable cachexia even in this stage, and marked loss of body weight, all point toward the correct diagnosis.

The condition of the remainder of the alimentary canal, particularly of the stomach, is of secondary importance. In discussing the objective symptoms we will describe the results of examination of stomach contents. Here we will only mention that, excepting during attacks, gastric digestion and appetite may be absolutely normal. If there be a long interval between the attacks of colic, the patients may gain so much in weight as to mislead one. This was particularly true of one of my own cases. In other patients —namely, those with increasing stenosis—the appetite very rapidly diminishes. This occurs partly in consequence of the frequent paroxysms of pain which rob the patient of sleep, and partly, according to Koenig, in consequence of auto-intoxication from the stagnant fæcal masses.

Objective Symptoms.—These are: tumour, meteorism, visible intestinal peristalsis, the character of the fæces, and, in some cases, the nature of the stomach contents.

The palpability of a tumour, which, according to Treves[30]*, occurs in 40 per cent of all cases of cancer of the large intestine, constitutes one of the most important and decisive diagnostic symptoms.*

The size of the *tumour* varies considerably, from that of a walnut to that of a fist, or even of a child's head. It is hard, nodular, incompressible, and more or less painful on pressure. According to all observers, its main characteristics are *active mobility*, and in connection therewith, *change of location and position.* The following limitations, however, must be remembered: the mobility of new growths of the large intestine is particularly well marked in those portions which, because of their long mesentery, themselves possess great mobility: these are the transverse colon with its flexures, and the sigmoid flexure. The cæcum and the ascending and descending colon, on account of anatomical peculiarities—namely, their short, tense, retroperitoneal mesenteries—are not nearly as mobile. Motion of the movable portions of the intestines may be restricted by adhesions. Passive and active mobility is increased by the weight and pressure of the stagnant intestinal contents above the tumour. At times the tumour is covered by distended intestine, so that it may be hardly palpable or disappear entirely; hence repeated examinations are necessary. This condition is very similar to that met with in carcinoma of the stomach, in which the tumour may not be felt when the stomach is full, but becomes palpable when that organ is empty. Another possible source of error is that the intestines themselves frequently change their positions, and thus the tumour may often be falsely localized. This subject, which is of great surgical importance, will be considered later. I shall now describe the general principles to be followed in the examination of intestinal tumours; the technic has already been considered (page 84).

Tumours of the large intestine are best palpated when the bowel is empty. When in doubt concerning the site of the tumour, or its differentiation from neighbouring organs (stomach, omentum, kidney, etc.), some aid may be derived from filling the intestines with air or water. As already stated (page 85), moderate inflation gives us much better information regarding the topography of the tumour and the position of the affected segment than extreme inflation. The latter procedure has its dangers in ulcerating tumours. In stenosing cancer of the lower portion of the large intestine, the fact that air or water *repeatedly* introduced *always* returns, may clear up an obscure diagnosis. *Meteorism* is another very important diagnostic symptom. It may be localized or general. When localized, it may enable us to determine the site of the stenosis; when general, the relations between the viscera become uncertain and obliterated. These symptoms are more thoroughly treated of in the chapter on Intestinal Stenosis.

The occasional occurrence of *visible intestinal contractions* ("intestinal rigidity," Nothnagel) is of great diagnostic interest. These contractions vary from a hardly noticeable rigidity of the intestinal coils to a plastic representation of one or more of these, and appear synchronously with the paroxysms of pain previously mentioned (page 307). Like these, they vary greatly in duration and occurrence. If contractions follow at frequent and regular intervals, their recognition is easy. If, on the contrary, they occur at long intervals, as in the beginning of stenosis, unless clinically observed, their recognition is mostly the result of accident. (See chapter on Intestinal Stenosis.)

The macroscopic appearance of the fæces may be of importance; its value, however, is negative rather than positive. The fæcal masses are generally not as long nor as thick as in normal stools. The stool is passed in small, pointed, narrow, or rounded masses. These characteristics are particularly noticeable in the stool from enemata. To this there are, however, many exceptions. I have seen an operated case of extreme stenosis from cancer of the cæcum, in which the calibre of the stool was absolutely normal. After passing the stenosis, the fæces must have become increased in calibre through additions.

The diagnostic importance of the passing of blood per anum has, I believe, been overestimated. In only 1 out of 11 cases of cancer of the large intestine did I observe blood in fairly large quantity. Slight losses of blood, usually overlooked, may occur more frequently. Treves[30], who has had much experience in these affections, has found hemorrhage in but 15 per cent of patients with cancer of the colon. Rüpp[4], in a series of 20 cases, has found this symptom present in 25 per cent.

As already mentioned, the dejections may be diarrhœal in character. This occurs not only in cancers of the lower segment of the large intestine, but, as soon as marked ulceration begins, also in those situated higher up. I have had two opportunities to make regular examinations of such stools. They are mottled red in appearance, and at times contain macroscopic, easily recognisable admixtures of pus, which sometimes forms as a yellow sediment, sharply contrasted with the remainder of the dejection. The evacuations were very foul, and always of a distinctly alkaline reaction. Besides red blood-corpuscles, microscopic examination showed many pus cells in every field, giving the impression of an abscess. There may be 8, 10, or 20 such dejections daily. Occasionally the pus may not be so prominent, and the stools are more bloody in character, but careful examination will always reveal pus in macroscopic or microscopic quantities.

As shown by cases of Potain[31] and Wunderlich[32], fragments of the tumour may very rarely be passed per rectum, a fact which will at once establish a positive diagnosis. In a case reported by Nicolaysen[33], there was prolapse of a carcinoma of the sigmoid flexure. These cases, however, are so extremely infrequent that as a rule they may be left out of diagnostic consideration. Despite careful search—both in spontaneously evacuated stool and in that after rectal irrigation—I have never found any tumour particles. Nevertheless, in all obscure diseases of the intestines in which blood and pus are present in the evacuations, I would advise frequent rectal irrigations for this purpose (see page 87).

The examination of the *stomach contents* may sometimes give facts of diagnostic importance. In cancer of the colon, as far as I know, no such examinations have yet been made. In three patients in whom I made the tests, the gastric motility was constantly normal. In one case there was absence, and in the two others an abundance of hydrochloric acid. I lay particular stress upon the maintenance of good gastric motility in cancer of the colon, since this, as is well known, suffers quite early in cancer of the stomach.

2. *Atypical Cases of Carcinoma of the Large Intestine*

There are many atypical cases in which the clinical picture of cancer of the large bowel is obscured; but, since their detailed knowledge may enable us to make a probable diagnosis, they must be considered. As in cancer of the stomach, there is often an entire absence of characteristic symptoms: the patient emaciates, loses appetite, presents indefinite dyspeptic symptoms and continues to lose strength, and finally dies. Autopsy shows an intestinal cancer. Such obscure cases occur in all segments of the gastro-intestinal canal. Surgical literature contains many examples.

In a second and fairly large variety of cases there is sudden, absolute intestinal obstruction with all its serious sequences. I believe these cases are more easily diagnosticated. As early as 1864 Bamberger[34] described one. After a meal consisting of lentils, the patient, till then an apparently strong, healthy man of forty, was suddenly attacked with severe abdominal pain, marked tympanitis, constipation, and vomiting. Death occurred on the third day. The autopsy showed a circular, carcinomatous stricture of the sigmoid flexure with only moderate stenosis, but the intestine above the tumour was entirely occluded by the undigested lentils. Rüpp[4] has described a series of similar cases coming under his own observation, characterized by acute intestinal occlusion from cherry stones, bone splinters, inspissated fæcal masses, and apple seeds. Very acute intestinal obstruction may also result from adhesions of the tumour to other intestinal coils, whereby kinking or twisting of the bowel is produced.

As exemplified by a case of Rüpp, impacted gallstones in the small intestine may cause the sudden appearance of acute symptoms of obstruction in a latent cancer of the large bowel. Such instances might be further multiplied. Even without such direct mechanical causes, symptoms of complete obstruction may (at least according to the patient's own statement) be induced by dietetic errors in healthy or rather, apparently healthy individnals. These acute attacks of intestinal obstruction are doubtlessly preceded by preliminary symptoms which are only slight, and impress the patient but little. As far as the patient's serious condition allows, the physician must inquire for symptoms which may have had the character of an incipient intestinal stenosis.

Differential Diagnosis

Without doubt the presence of a well-defined tumour generally facilitates the diagnosis; although even then error may be unavoidable.

The question whether the tumour is a real neoplasm or only impacted fæces, may sometimes produce the greatest diagnostic difficulties. In the general division (page 76) we have described the various methods by which mistakes may usually be avoided. We have there stated that fæcal accumulations above the stenosis may make a neoplasm appear much larger than it really is.

Tumours of the cæcum, beginning like appendicitis, with fever, pain on pressure, and resistance in the ileo-cæcal region, often lead to diagnostic errors. But these tumours do not disappear when the acute symptoms have passed; they become larger and more nodular, the patient emaciates, and dies in extreme marasmus, or he may succumb to symptoms of acute intestinal obstruction. Several such cases have been described by Bamberger[35] and Krausshold[36]. Schede[37] has observed a medullary cancer superimposed upon an old, irregular perityphlitis. On the other hand, Schede and Richelot and Hartmann[38] have operated on cases in which, instead of an expected cancer of the cæcum, they found only inflammatory perityphlitic products. These cases demonstrate that, even where a tumour can be felt there may be difficulties.

If there be an intestinal tumour the further question of malignancy or benignancy will have to be determined. Regarding benign tumours, fibromata and myomata of the intestines are very rare, and only exceptionally produce the severe symptoms of malignant growths. The differential diagnosis between sarcomata of the large and small intestines may also have to be made. It is discussed under the heading Symptomatology, to which the reader is referred.

We have already discussed the interesting and frequent question of differentiation between cancer and tuberculosis of the cæcum (p. 277).

Besides the difficulties already mentioned, tumours of other organs may give rise to errors in diagnosis. For example, von Bergmann[39] once diagnosticated a tumour as cancer of the cæcum; on operation it was found to be a cancer of the stomach adherent to the right iliac fossa. Hahn[40] reports a case of a young man of nineteen with a nodular tumour in the right side which was thought to be an enlarged kidney. Laparotomy (Simon's incision) showed the right kidney normal and in normal position. Upon opening the peritoneum there was found a tumour of the ileum and cæcum the size of a child's head. Examination proved it to be a small round-celled sarcoma. Czerny[41] and von Esmarch[42] report similar errors. In a doubtful case in a woman, in which the diagnosis rested between tumour of the cæcum and floating kidney, Salzer[43], on vaginal examination, was able to differentiate by involuting the soft layers between his fingers and the tumour; he could introduce his fingers into the ileo-cæcal opening from the small intestinal side, and thereby recognised the neoplasm as one of the cæcum. These examples, particularly abundant in surgical literature, might be multiplied.

The diagnostic difficulties are increased when tumours of the intestine (generally the large intestine) are complicated by displacement of the different intestinal segments. The obstacles are so great that sometimes even an operation will not clear up the cases. Thus, Pässler[44] reports a case from Curschmann's clinic in which a carcinomatous stenosis of the hepatic flexure of the colon was clinically diagnosticated. Autopsy revealed a carcinomatous degenerated cæcum, which was situated high up under the liver, and, owing to a congenital absence of the ascending colon, communicated directly with the transverse colon. During an operation for carcinoma of the large intestine, Israel[45] thought he was dealing with the descending colon, while, in reality, he was operating upon a displaced transverse colon.

Many more examples might be mentioned. They should teach us to be very cautious in diagnosticating the site of a neoplasm. It may be necessary to differentiate between cancer of the large intestine and *chronic intussusception*. The following symptoms point to intussusception: sudden onset, passing of blood per rectum, shape of the tumour (smooth, cylindrical) and its spontaneous motility, the age of the patient. None of these symptoms is pathognomonic, and only by careful consideration of the separate data can a mistake in diagnosis be avoided. Diagnostic difficulties may become considerable when a tumour can not be palpated, and when the usual characteristic symptoms are absent. However, when there is a good clinical picture a probable diagnosis can be made. To begin with, it is necessary to establish the existence of an intestinal stenosis. If the symptoms of the latter are well marked, we may generally arrive at the correct diagnosis by exclusion. Aside from subjective symptoms, it is necessary to keep in mind, first of all, the appearance of visible intestinal peristalsis; one single coil of intestine discovered in the act of peristalsis and rigidity may clear up an otherwise doubtful condition. For the different symptoms which occur in the several varieties of intestinal stenosis I refer the reader to the chapter on Intestinal Stenosis.

If intestinal stenosis is absent as a symptom, the diagnosis is only possible from the presence of other objective signs, particularly blood and pus in the stools. As Nothnagel * states, the only other disease besides cancer which can come into question when bloody, purulent matter is found in the dejections, is chronic dysentery. "Since dysentery is generally easily recognised, the importance of this type of dejection in the symptomatology of intestinal cancer is quite manifest."

The differentiation is not always easy, as the following example will show:

Mr. A., merchant, thirty-nine years old, from Hanover. Parents and grandparents died at an advanced age; has one brother alive and well. Had measles when a child. When thirteen years old had malaria (probably tertian type) for eight to nine weeks, later disappearing entirely. At twenty-four had hemorrhoids, which were successfully ligated. Otherwise the patient was well until 1896. At that time he suffered with bowel complaint—frequent tenesmus without evacuations, abdominal pain, alternating constipation and diarrhœa. Blood or pus had not been observed in the stool. There was then no marked disturbance of general health; the patient was not confined to bed; he was able to attend to his business. Following the use of hot enemata the stools regained their normal consistency, and remained regular until November, 1897. Patient then for the first time noticed the occurrence of frequent painful rectal tenesmus. The dejections were liquid, had a very bad odour, and were mixed with blood and mucus. The tenesmus and number of dejections gradually increased. Patient began to have fever; he emaciated and lost his previous good appetite. I first saw him on February 10, 1898; the *status præsens* was as follows: Extremely pale, emaciated man, of medium size and cachectic facies. No enlarged glands, no œdema, no exanthema; tongue dry, clean, red; throat showed nothing special; thorax long and narrow, percussion note normal; vesicular breathing present all over the lungs. Heart sounds normal; heart of normal size. Pulse of very low tension, small, somewhat irregular, 120 beats per minute. Temperature, 36° to 37° C.

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* Loc. cit. (ref. 6), p. 236.

Abdomen.—The entire abdomen unequally distended. There is distention, particularly localized below the umbilicus. Occasionally there are seen indications of intestinal peristalsis without rigidity, particularly in the ileo-cæcal region. No tumour can be felt. Palpation of the ileo-cæcal region is very painful. Hepatic and splenic dulness entirely absent, being obscured by the tympanitic intestinal percussion note. Slight splashing sounds in the epigastrium. Rectal examination negative. Rise of temperature between 38° and 39° C.

Since his stay in Berlin the patient has had continual rectal tenesmus. There are 6 to 8 stools daily, consisting at first mainly of pus and blood, with only a small amount of fæcal matter; they have a very foul and fetid odour. Microscopical examination shows innumerable large and small pus cells and blood cells. No amœbæ. Repeated examinations for tubercle bacilli are negative. Pus in the stool continues during the course of the disease.

Urine contains a moderate amount of indican. Quantity of urine, 500 cubic centimetres in 24 hours; it is brownish-red and of high specific gravity.

The course of the disease was as follows: There was quite apparent loss of strength; at first fever, later temperature is normal or subnormal. Despite this the pulse is always 120–130, and very small; tongue clean but dry; absolute anorexia, troublesome thirst; the main subjective symptom is tenesmus; the main objective symptoms are meteorism, pain in the ileo-cæcal region, purulent dejections, as well as the patient's general septic condition (septic intoxication). With symptoms of increasing marasmus and occasional somnolence, the patient died, February 26, 1898.

Autopsy, February 27th, performed by Dr. Hans Kohn, of Berlin. Very much emaciated corpse, presenting nothing special externally. *Abdomen* moderately tympanitic. The thin abdominal walls coloured green. Upon opening the abdomen all the intestines were found to be distended with gas, and were all in normal position except the sigmoid flexure, which lay parallel with the pubic bone until it reached the right iliac fossa, where it was slightly adherent. It was also adherent to the anterior circumference of the pelvic outlet. The omentum was almost entirely free from fat and wreathlike in shape, and also adherent to the pelvic outlet. After the omentum is thrown back the general intestinal serosa is moist and pale, excepting over the sigmoid, where it is very dark red. Attempting to loosen and free the sigmoid, the whole intestinal wall easily tears, disclosing an abscess cavity filled with pus and about the size of a hen's egg. The walls of the cavity are mainly formed by the sigmoid flexure, and partly by the anterior pelvic floor. It communicates directly with the lumen of the bowel; the size of the communication can not be positively determined because of the extremely brittle condition of the intestinal wall. The dark discoloration of the intestinal serosa extends upward to about the beginning of the descending colon, whence it gradually becomes pale and disappears. With the exception of the beginning of the ascending colon, which is filled with fæcal matter, the large intestine is found to be empty, or rather to contain only gas. The mucous membrane of the descending colon is pale yellowish-white in colour, and is uniformly covered with thick pus. In numerous places there are ulcerations, irregular in shape, with smooth edges, and extending to the muscularis. Above, the ulcers may be followed into the middle of the transverse colon, where they become smaller. The smallest are the size of lentils, the largest the size of a 50-cent piece. They are not round, but irregularly shaped. Below, the ulcers increase in size, and the mucous membrane becomes gradually more deeply injected. In the sigmoid flexure the greater part of the mucous membrane is destroyed. Here there are areas of eroded mucous membrane about 1.2 centimetres long, 1 to 2 centimetres broad, and about 1 to 3 centimetres thick.

As just stated, areas of mucous membrane and muscularis are entirely destroyed. Between these the mucous membrane is covered by reddish-yellow thick pus. These changes extend into the rectum. The mucous membrane of the upper portion of the large intestine, and of the whole of the small intestine, is anæmic. The small intestine contains only small quantities of semisolid masses; the contents of the cæcum are of normal consistency, formed, rich in fat; but in the middle of the ascending colon the contents become semisolid. As already remarked, the intestines situated farther down are empty.

Isolated lymph nodes the size of lentils are found in the walls of the lower segments of the large intestine. Some of the mesenteric glands are swollen to the size of beans. There is no thrombosis of the blood-vessels supplying the descending colon.

The liver is quite small and soft; on section it is pale, reddish-yellow, and cloudy; it contains no abscesses.

The spleen is somewhat enlarged, bluish-red, and soft.

The right kidney, normal in size, but soft and grayish-red on section; very cloudy.

The clinical diagnosis lay between carcinoma, tuberculosis of the large intestine, and chronic dysentery. Since tubercle bacilli were never found, and since other signs of tuberculosis were absent, the diagnosis was limited to the two other possibilities—carcinoma and dysentery.

In my opinion, an epicritical examination of the case presents no possibility of a positive differentiation; as against cancer it might be maintained that a tumour was absent. But a tumour, if present, would have been obscured by the marked abdominal distention. As regards the fever, that would have spoken as much for the one as for the other condition. As to the age of the patient, there is no special limit within which cancer may occur, particularly cancer of the large intestine. Finally, the course of the disease and its acute invasion spoke rather for than against a malignant neoplasm.

I might describe an analogous case, very similar throughout except that toward the end of life there developed to the right of the bladder a tumour, whose diagnosis caused expert clinicians, as well as myself, many difficulties. These difficulties were increased by the fact that the patient dated his symptoms some fifteen years back. Operation showed a sloughing carcinoma of the sigmoid flexure, which had displaced the latter to the right.

From these two cases it follows that, in the absence of a tumour, the differential diagnosis between dysentery and carcinoma may cause great difficulties, which, so far as I can see, cannot, in the present state of medical knowledge, be overcome.

Finally, cancer is to be differentiated from the *intestinal neuroses.* I have observed two cases which for a long time presented symptoms of nervous intestinal disturbance, and whose malignant character was revealed only late in the disease. Both patients had suffered for years from habitual constipation, and both were marked hypochondriacs on the subject of defecation. Contrary to my own opinion, and quite correctly, they looked upon their last complaint as of a very serious nature. Brinton's excellent dictum regarding cancer of the stomach is also true of cancer of the intestine: "Obscure in its symptoms, frequent in its occurrence, fatal in its events."

(*c*) CANCER OF THE RECTUM

Cancer of the rectum is recognised more easily than cancer of any other portion of the intestines, and offers the most favourable chances for cure.

Symptomatology and Diagnosis

It is best not to separate the subjective from the objective symptoms, but to consider them together. The subjective symptoms relate to the disturbances of defecation, and at the beginning they may be so indefinite that the patient may not seek medical advice.*

Defecation is interfered with; evacuation occurs only after strong action of the abdominal muscles, and the stools have no longer the normal cylindrical form, but are flattened and of small calibre.

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* Occasionally apparently remote symptoms may point to the real source of trouble—e. g., obstinate sciatica.

They resemble sheep dung, and are often fragmentary. It thus happens that patients have frequent daily evacuations, but each time these are small, unsatisfactory, and are accompanied by very much straining. Closely related therewith is a feeling of fulness, weight, and pressure in the small pelvis, which always impels the patient to attempt to evacuate his bowels. The resultant movements, though small, afford temporary relief.

Gradually painful tenesmus develops, together with the increasingly frequent and scanty dejections. These may remain formed, but usually consist of thin fluid masses with an exceedingly nauseating, fetid odour. At this time the dejections may contain mucus, blood, and pus.

As the disease progresses the symptoms of a stenosis of the rectum become more marked; tenesmus is continuous, or has only short remissions. The stools become more numerous, more liquid and less in quantity, and admixtures of pus, mucus, and blood are more often found. The symptoms continue during the night and cause insomnia. The appetite decreases perceptibly, the general health begins to fail, and the patient begins to look cachectic.

The objective examination consists, first and foremost, of a digital exploration of the rectum, and then of an examination of the evacuations.

At varying distances from the anus the examining finger encounters irregular, nodular, thickened masses which are immediately recognised as neoplasms. A more careful examination will distinguish two types. In cancers situated *high up*, the finger has the feeling as if entering a hard, rigid cylinder, above and to the sides of which is attached, as it were, a vaginal vault. Generally, the finger cannot pass any farther through this pseudo-vaginal opening; with a little force it may enter a narrow irregular cylinder. On withdrawing the finger there are traces of blood and a characteristic fetid odour.

Kraske[9] states that where the carcinoma is limited to the wall of the rectum, and is not adherent to the surrounding tissues, one may obtain the sensation of ballottement with the end of the finger. I have found this symptom only once, but this infrequency is presumably due to the fact that I generally see cases in the more advanced stages of the disease.

In cancer situated *low down* in the rectum there is usually no marked invagination. The finger enters a stiff walled cavity which is sharply defined against the smooth mucous membrane, both above and below. In other cases there are circular or semicircular tumours, with protuberant, serrated (cockscomblike) edges which project beyond the normal mucous membrane.

The diagnosis of the presence of cancer of the rectum is not sufficient; it is also necessary to know the extent of the tumour, its mobility, and the presence or absence of complications. The question of mobility is of the greatest surgical significance. According to Kraske, the question of operation depends more on the mobility of the growth than on its size.

The main complication is rupture into the neighbouring viscera (bladder, genitals). We will not discuss the rarer complications here.

In every case of rectal carcinoma the *liver* ought to be examined as a matter of routine. Metastases occur most frequently in this organ, a fact which is naturally of great importance in the question of radical operation. Ordinarily the examination of the fæces is unnecessary; but in doubtful tumours, or in those reached with difficulty, such an examination may, as I maintain in opposition to Hochenegg[46], clear up the clinical picture to a considerable extent. The external appearance of the fæces may either be that of stenotic stools (e. g., ribbonlike, spiral, short cylinders embedded in a thin, apparently homogeneous, bloody, or purulent ground substance), or they may consist entirely of fluid or semifluid masses, or, in advanced cases, of pus and blood. In cases not far advanced, where the cancer is situated high up or where the differential diagnosis lies between cancer and benign neoplasm, the abnormalities of the fæces just described are of less significance than the demonstration of small, microscopic admixtures of pus.

The diagnosis, therefore, will not be difficult in the majority of cases, particularly to the physician who makes it a rule to examine the rectum digitally not only in patients with symptoms referred to the rectum, but in every case of intestinal disturbance. There are, however, isolated instances in which there will exist doubt regarding the nature of the rectal affection. These necessitate a short discussion on differential diagnosis.

DIFFERENTIAL DIAGNOSIS

By careful and repeated rectal and vaginal examinations it is very easy to differentiate tumours of the rectum from tumours of the prostate or of the female genitals, pelvic abscesses, etc. Polypi of the rectum will only exceptionally cause diagnostic difficulties. Owing to their extreme rarity, myomata of the rectum will scarcely come up for consideration. Differentiation from rectal sarcoma may give rise to error; in contrast to cancer, the sarcomatous tumour has a smooth surface and there is no tendency to ulceration.

It may be somewhat more difficult to distinguish between fibrous syphilitic stricture of the rectum and carcinoma. We shall more fully discuss the symptomatology of syphilitic stenosis of the rectum in the chapter on Diseases of the Rectum, and therefore limit ourselves here to a few brief remarks.

With Kraske, I believe that the differentiation is not difficult. With reference to the diagnosis, Kraske says the following, which corresponds with my own experience: "In syphilitic proctitis the stenosis is produced by cicatrization and is a real stricture. The difference between the two forms of stenosis is also very evident to the examining finger. The syphilitic ulcerations never have the hard, protuberant edges that are found in the carcinomatous. In contrast to cancer, syphilitic ulcers are generally multiple, and are separated from one another by areas of healthy or cicatrized mucous membrane. The syphilitic infiltration begins mainly as a diffuse process, while cancer is for a long time more circumscribed. This last fact is particularly evident in the condition of the surrounding tissues and organs. Syphilitic ulcerations very often produce periproctitis, external abscesses, and fistulæ which rupture externally, while this very rarely occurs with cancer. In my own experience it has never occurred. It is true that in cancer situated low down there may occasionally be a rupture through the skin in the neighbourhood of the anus, but the character of such an opening, particularly its infiltrated margin, will at once show that it is not a fistula arising from a periproctitic abscess, but is a direct rupture due to the growth of the cancer toward the surface." If we add further that syphilitic stenosis is essentially chronic, is more frequent in women than in men, is present much earlier in life than cancer, we have sufficient facts to assist us in most cases. Where, despite the above, diagnostic difficulties are encountered, there is the final recourse to excision of a piece of the growth for examination. Even this may not give positive results.

COMPLICATIONS OF INTESTINAL CANCER

The most important complications are produced by the tumour itself. As already mentioned (page 300), the tumour may become adherent to the bladder, uterus, ovaries, stomach, etc. There may be a discharge of fetid or fæcal matter through these organs. I have seen two cases of rupture of cancer into the bladder with the rapid development of a purulent cystitis with extremely feculent urine. When rupture into the uterus or vagina takes place, fæcal masses empty themselves through the genital cloaca. Where a communication between the stomach and large intestine is established the condition of lientery, known to the older writers, develops. In consequence of such a fistula there is fæcal vomiting, and the passing of entirely undigested food per anum. Rarely, carcinoma may rupture through the abdominal wall. Finally, there may be a rupture of the carcinoma into the retroperitoneal tissue, with the formation of fæcal abscesses. The latter may cause a general septic peritonitis, or may lead to abscesses pointing at different places—e. g., Poupart's ligament, the lumbar region, etc. The perforation, per se, is practically the most important as well as the most significant complication. This may occur very suddenly, without any warning, when the patient seems to be improving. In one case perforation occurred during the time the attendants were giving the patient an enema of water. Straining at stool may cause this unexpected accident.

Finally, death may be caused by rarer complications, viz., aspiration pneumonia, embolism and venous thrombosis, uræmia, metastases in other organs, peritoneal carcinoma, terminal hemorrhage of the intestine, stomach, etc.

TREATMENT OF INTESTINAL CANCER

In the ordinary sense of the word a real cure of an intestinal cancer does not exist. Under favourable conditions, to be later more fully described, extirpation of the tumour may prolong life for months, or years, but even in these cases a fatal termination cannot be prevented. With but few exceptions, surgical interference is the best and most practical of all the palliative, life-prolonging remedies. In most surgical operations there is undeniably a direct relation between the object to be gained and the severity of the operation. He who risks much may occasionally expect a successful result even under unfavourable circumstances; he who does not take such risks cannot reckon on great results.

In hopeless cases we must limit ourselves to palliative treatment. The various palliative measures depend upon the site of the tumour and the clinical syndrome.

They consist in

1. Increasing the patient's strength.
2. Removal of the stenotic symptoms present in the great majority of cases.
3. When the last object is only partially accomplished or impossible, relief of the pain and other symptoms caused by the stenosis.
4. Treatment and relief of complications.

It is quite apparent that the above division is somewhat schematic, since the several symptoms may change or become interdependent. For the better survey of the subject we shall, however, adhere to this grouping.

1. In many cases the attempt to increase the strength and nutrition succeeds even though temporarily. For apparent reasons we are least successful in cancer of the small intestine, and most successful in cancer of the rectum, while cancer of the large intestine occupies a middle ground.

In cancer of the *small* intestine the diet is similar to that in cancer of the stomach. It consists in the frequent administration of small quantities of fluid or semisolid nourishment of the highest caloric value. To stimulate the appetite we must consider the wishes and peculiarities of the patient. We should not hesitate to give patients such food as may refresh and please them, provided it does not aggravate the intestinal lesion (von Leyden).

The underlying principles have already been given (page 151), and it will only be necessary to describe several minor details. In carcinoma of the duodenum those foods are most appropriate which, because of their physical character, allow of a large concentration of soluble nourishment. Milk, albumin, carbohydrates, and fats, percentages of which may be increased at will, occupy the first place.

The albumin may be increased in amount by the addition of commercial albumin preparations; the carbohydrates, by the addition of flour in any of its many well-known forms; the fats, by addition of cream in amounts depending upon the tolerance of the stomach and the degree of stenosis. Both the albumin and carbohydrates can be simultaneously increased by adding leguminous flour.

The artificial albumin preparations, often tiresome in dyspeptic conditions, may be alternated with natural egg albumen. Vegetable, meat, and fish soups in their many combinations may be given to satisfy the patient's desire for change of diet without producing an appreciable diminution of the general nutrition. Solid meat and fish preparations should be administered in their most easily digestible forms, or had better be avoided altogether. Vegetables and fruits, white bread, zwieback, sweetened crackers, all of which have been finely divided or thoroughly cooked, may be allowed. Raw fruits, vegetables, tubers, and similar articles are to be absolutely forbidden. We shall return to this subject.

The food given to patients with *cancer of the rectum and other portions of the large intestine* may be much more varied. As we have already seen, the stomach functions may be absolutely normal in these cases, and a similar normal condition may be assumed to exist in the upper part of the intestinal canal. The diet must be of the greatest caloric value, but the food need not necessarily be given in small quantities and with frequent intervals. With the exceptions soon to be stated the foods may be given in their natural form. Meat, when minced, is often easily digested. Soups, particularly when concentrated, are good though by no means absolutely necessary forms of diet. The same may be said of milk, though strengthening the patient. Unfortunately milk is not always well borne; in such instances it may be tried in its various preparations (kefyr, sour milk, koumyss, etc.).

Vegetables, *which in their natural form are not finely divided, must always be strained, so as to prevent any possible mechanical obstruction of the lumen of the intestine.*

For this reason vegetables which cannot be mashed should be absolutely excluded; in fact, I regard it as a distinct therapeutical error to allow patients with intestinal stenosis to eat unstrained lentils, peas, beans, asparagus, raw fruit, cabbage, etc. This same is true, possibly more so, when applied to raw *compotes*, or such as are not rendered fully pultaceous, or *compotes* and fruit containing small seeds. As shown above, the patients may pay for such indiscretions with their lives.

2. Next to abundant nourishment, the physician must try to

induce increased intestinal peristalsis, brought about, if possible, by dietetic means. We have fully described this in the General Division (see page 151, etc.).

To a certain degree diet may remove the symptoms due to the stenosis. The underlying dietary principles have already been considered in the General Division, and in the chapter on Intestinal Stenosis. The functional disturbances from intestinal cancer require great caution, for to the putrefaction produced by the stenosis there is often added that resulting from the intestinal ulcerations. Thus, instead of the normal contents, the bowel contains a fetid mixture, uninfluenced by intra-intestinal medication. It is advisable to get rid of this putrescent material as soon as possible by lavage of the stomach in cancer of the upper intestinal segments, and by appropriate laxatives when the cancer is in the lower segments of the intestine.

3. When the above measures fail to relieve the symptoms of stenosis, the use of narcotics is oftentimes unavoidable. We must again refer the reader to the chapter on Intestinal Stenosis and to the General Division.

In the *operative treatment* of intestinal carcinoma the following indications are particularly to be considered:

1. Extirpation of the tumour.
2. The removal of stenotic symptoms.
3. Relief of intestinal obstruction which may develop during the course of the disease.

Before discussing the indications, we must briefly narrate the results of intestinal surgery, as found in the numerous clinical and statistical reports.

The medical practitioner, even though thoroughly acquainted with the various phases of the disease, will rarely have an extensive personal experience therewith. Hence, notwithstanding the well-known and oft-repeated fact that statistics are not always complete or reliable, it is necessary for him to study statistical reports.

In the first place, the results of operations for cancer vary with the kind of operation. According to Wölfler[47], the latest statistics on intestinal resection for new growths give the high mortality of 54 per cent, while the entire mortality of intestinal resections for all causes is only 39.5 per cent. As pointed out by many writers (Mikulicz and others), this mortality is influenced by the nature of the disease. In this connection it is worthy of notice that better technic shows no improvement in results. In

1890 Billroth[48] reported a mortality of 50 per cent; Czerny[49], a similar mortality in 1892. Nicolaysen[50] gathered together 121 cases from literature, with a mortality of 48 per cent. My own experience is limited to 8 operations for cancer of the large intestine (cæcum, 5; hepatic flexure, 2; sigmoid flexure, 1), of which 3 were resected. One case, cured by resection, is now alive, four years since operation; 2 died several days after operation; intestinal anastomosis was performed in 2 cases (they lived eight or nine months); in 2, enterostomy (colostomy) was performed during an attack of acute intestinal obstruction; in another, exploratory laparotomy.

The chances for a permanent cure after resection, are evidently more favourable in intestinal than in gastric cancer, because the former has a lesser tendency to metastasis. Wölfler reports a case of a man operated on for cancer of the sigmoid flexure in 1879 who was still well in 1896. Rüpp[4] reports a case of a patient from Krönlein's clinic, who after nine years had had no relapse. From reports kindly sent me by Prof. Körte, of Berlin, I learned that an almost hopeless case of cancer of the cæcum had been operated on and had now been well over nine years. There are many other similar accounts of patients operated on for cancer of the large intestine who remained cured for a number of years (Billroth, König, Czerny, Wölfler, and others).

The results from incomplete enterostomy (the entero-anastomosis of Maisonneuve) are much more favourable. Wölfler[47] cites statistics of Schloffer, which show that in 47 cases of intestinal stenosis the mortality from this operation was only 30 per cent. The duration of the cure seems on the whole to be favourable. In one case of Körte* (cancer of the splenic flexure) the patient lived three and a quarter years after entero-anastomosis; he died finally of metastatic cancer of the liver. The result in these cases is to be judged by the degree of restoration of function. Unfortunately there are only a few useful reports in this connection. In the two cases I have mentioned above the results were not satisfactory. The patients gained in weight, although the intestinal pains were in no wise decreased. In one of the cases evidences of intestinal stenosis reappeared after a time.

The results of operative treatment of rectal cancer require special mention. We distinguish the following methods:

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* Personal report.

1. Extirpation of the rectum by the perineal method, practised in rectal tumours situated low down.

2. Extirpation by the sacral method, first introduced by Kraske[9] for rectal cancer situated high up; the operation has been improved by the further modifications of Hochenegg[51], v. Heinecke[52], W. Levy[53], Schlange[54], [Bardenheuer] and others.

3. The vaginal method recently introduced by Rehn[55] for the removal of rectal cancer in women.

There are a number of statistics regarding the results from the first of the above-mentioned surgical procedures. We limit ourselves to the very extensive statistics from Czerny's clinic[56], which have the advantages of presenting the results of only one individual, of covering a long period of time, and of including both methods of operation. From 1878 to 1891 152 cases of rectal cancer came under observation, of which number radical operation was performed in 109, 21 were curetted, 12 were inoperable, and colotomy was performed in 8. Of 83 cases operated by the perineal method, 3 died immediately (that is, 3.6 per cent). Of 66 cases operated on by the sacral method, 9 died (13.64 per cent). The total mortality of the 109 cases was 10, or 9.1 per cent. Of 99 patients radically operated on, 21 lived two years or more; 15 lived three years or more; 8, five years and over; 1, eighteen years; another, sixteen years; others, thirteen and three quarters, eleven and a half, eight and three quarters, and six and three quarters years. The frequency of recurrence after extirpation is very variously estimated by the different authors. The percentage ranges between 41.6 per cent (Kraske) and 73.3 per cent (Lövinsohn). According to Czerny[57] these figures underestimate the facts; he claims that 20 to 25 per cent of radically operated cases remain free from recurrence for over two years, and that the majority of these remain permanently cured. The danger of recurrence is diminished by the sacral method of operation, since by this procedure the lymph nodes in the sacral fossa can also be removed. But the value of extirpation of the rectum is also determined by the functional results obtained. Unfortunately, there are not sufficient statistics in this connection to permit of proper judgment. The functional results depend upon whether the sphincter ani must be sacrificed or not. When the sphincter can be saved the functional result is satisfactory, even though the sphincter rarely contracts as well as the normal. When the sphincter has been sacrificed, the condition of the patients is extremely unfortunate, since they have absolutely no control over the flatus or fluid stools. Solid fæces can usually be controlled by the formation of an elastic obstruction near the former third sphincter, the rectum being then daily irrigated.

Regarding extirpation of the tumour, whether of the small intestine, colon, or rectum, the operation must be radically performed in those cases in which the tumour is well circumscribed and movable, and where no metastases are found.

As is well known, apparently favourable cases may, when laparotomized, present evidences of metastases, ascites, and peritoneal carcinoma—conditions which make radical operation illusory. When an early diagnosis has been made, it is inadvisable to delay operation, for, aside from favourable local conditions, resection of the intestine requires endurance and strength on the part of the patient. Unfortunately, the absence of these qualities frequently renders radical operation impossible.

The second indication for operation is increasing stenosis. In these cases a radical operation cannot be performed, either because adhesions make the removal of the tumour very difficult, or metastases are already present, or the weakened condition of the patient does not allow of a severe operation. In cancer of the small intestine gastro-enterostomy or entero-anastomosis comes in question; in cancer of the large intestine (including the sigmoid flexure), entero-anastomosis or colostomy; while in cancer of the rectum only colostomy. In rectal cancer some surgeons advise scraping or electrolytic removal of the stenosing tumour masses, but others of experience (among them Kraske and Czerny) advise against such procedures.

When there is danger of intestinal obstruction or when it is already present, enterostomy or colostomy is generally indicated.

Finally, regarding extirpation of rectal cancer, the indications to operate are influenced more by the mobility of the tumour than by its extent. In many cases examination under narcosis is necessary to decide the question of operability and the character of the surgical procedure to be used. In all obscure cases it is best for the medical practitioner and surgeon to consult, and together determine the mode of operation.

The experienced medical practitioner can generally recognise inoperable cases of cancer of the rectum; the extensive, rigid, fissured, ulcerated, absolutely immovable neoplasm leaves no room for indecision. In these cases we must decide whether a colostomy is to be performed, or whether the patients are to be left to their fate. The decision is by no means an easy one; each individual case must be carefully considered, not only respecting the condition of the rectum, but also the personality of the individual. Considering the unfavourable cosmetic result, some surgeons strongly advise against colostomy, and only resort to the operation when an almost complete stenosis is present.

Kraske[9] says: "It is horrible, even for a person of phlegmatic temperament, to witness daily the misery caused by the involuntary evacuation of fæces, and to know besides that a progressive and fatal disease is present." Frequently, however, patients are satisfied with their condition after operation.

II. Sarcoma and Lymphosarcoma of the Intestine

According to Kundrat[58], the best authority on these neoplasms, sarcoma and lymphosarcoma belong to the rarer intestinal tumours. Out of the large material of the Vienna General Hospital, he could collect only 3 sarcomata and 9 lymphosarcomata of the intestine between the years 1882 and 1893.

Regarding the localization of sarcoma, Nothnagel * voices the general opinion when he states that the majority of cases are found in the small intestine, and only extremely rarely are they in the large intestine.† This view is correct if we exclude the rectum, where sarcomata are found as frequently as in the upper part of the intestinal canal. This conclusion is drawn from the careful work of Fr. Krüger[59], who has tabulated all the known cases of intestinal sarcoma reported up to the year 1894—altogether 37. These tumours were distributed as follows:

Small intestine	16
Ileum and cæcum	1
Cæcum	1 (2) ‡
Vermiform appendix	1
Transverse colon	1
Small and large intestine	1
Rectum	16

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* Loc. cit., p. 250.

† [Libman, American Journal of the Medical Sciences, September, 1900, p. 309, publishes an interesting report of four cases with detailed discussion of the clinical and histological aspects of sarcoma of the small intestine, together with an extensive reference to the literature of the subject.—Tr.]

‡ A case reported by Carrington (cited by Baltzer, Archiv für klin. Chir., vol. xliv, p. 744) has here escaped notice.

The ages of the patients were as follows:

3 cases in the first decade.
3 " " second "
6 " " third "
10 " " fourth "
5 " " fifth "
6 " " sixth "
4 " " seventh "

If we may judge from these rather few statistics, we see that the first decades by no means furnish so large a majority of the cases as is commonly believed. Furthermore, in contrast to cancer, the proportion of the male and female cases is striking (31 : 6).

Regarding the type of intestinal sarcoma, all possible forms may be observed—hard and soft, small and large spindle-celled, small and large round-celled, alveolar and medullary, melano- and cystosarcoma (Krüger). The round- and spindle-celled forms* are the most frequent. They generally originate in the submucosa, extending inwardly to the tunica propria and outwardly to the muscularis. Some forms originate in the subserosa, and spread inwardly from here.

The sarcomatous tumour is usually smooth and its size varies. It is often enormous. At different places it presents softened areas. It is not particularly painful, is frequently movable, and, in contrast to cancer, is distinguished by its rapid growth.

Lymphosarcomata probably originate from the lymphatic system of the intestines. As already indicated by the difference of their development, the changes produced in the intestinal canal by sarcomata and lymphosarcomata are quite different from those of cancer.

In cancer there is a circular, relatively well-defined area of disease; in sarcoma and lymphosarcoma the affected area is extensive and indefinite. As a result of this circular form of the cancerous tumour a stenosis results. In sarcoma, however, as pointed out by Treves, and later also by Baltzer[60] and Madelung[61], there is almost always more or less demonstrable stretching and dilatation of the intestinal lumen. As demonstrated by Bessel-Hagen[62] in a case he reported, the dilatation may become enormous. Sarcoma is much more apt to form metastases than is cancer, but, on the whole, this occurs late in the disease. Lymphosarcoma, on the other hand, seems to attack only the surrounding lymph glands.

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* For further histological details I would refer to the text-books on pathological anatomy; also particularly to the article by Ackermann, Die Histogenesis und Histologie der Sarcome, Volkmann's Samml. klin. Vorträge, pp. 233, 234.

The question of the relation between tuberculosis and lymphosarcomata is a peculiar one and has recently caused lively discussion. The observations of A. Müller[63], Claus[64], Ricker[65], Nothnagel,* Dietrich[66], and Rud. Schmidt[67], point to the possible coincidence of tuberculosis and lymphosarcomatosis.

Hereditary tuberculosis is sometimes found in the family history. A direct connection between the two diseases seems to be excluded; still there is much in favour of the theory advanced, particularly by Rudolf Schmidt, that there probably exist hereditary constitutional tendencies—that is, a sort of lessened power of resistance of the entire lymphatic system.

The duration of intestinal sarcoma is shorter than that of cancer; most patients die within one year of cachexia, metastases, etc.

SYMPTOMATOLOGY, DIAGNOSIS, AND DIFFERENTIAL DIAGNOSIS

The *subjective symptoms* of sarcoma of the small intestine present but few characteristic features. They consist in diffuse abdominal pain, nausea, vomiting (oftentimes said to be bilious in character), and marked irregularity of the bowels—obstinate constipation alternating with profuse diarrhœa. Where constipation alone was present, complications were always found (invagination or intestinal displacements). The most important *objective symptoms* are the tumour, intestinal symptoms, and rapidly developed debility. In contrast to cancer the tumour is smooth, and often softened areas are to be felt; it is well defined and easily movable. Several cases have been reported (Madelung) in which there was evident growth of the tumour within a few days.

As is apparent from the above, the absence of the symptoms of stenosis is a characteristic of great diagnostic importance; visible intestinal peristalsis and intestinal impactions are also absent. On the other hand, the extensive growth of the sarcoma may cause severe intestinal paralysis.

To judge from a review of the histories of reported cases, the absence of intestinal hemorrhage is of some diagnostic significance, since this symptom is rather frequent in cancer. The very rapidly developed cachexia is a striking symptom; it may become extreme within a few weeks or months.

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* Loc. cit., p. 253.

Other symptoms of possible diagnostic value are temporary *œdema of the ankles*, the early occurrence of ascites, and occasionally an irregular fever (up to 39.5° C. in a case of Madelung's). I find œdema of the ankles mentioned as a symptom in six of Krüger's[59] cases.

The recognition of lymphosarcoma is very difficult, for, as shown by a study of the cases published, its symptoms may be quite different from those of sarcoma. Two examples of this are reported from Neusser's clinic in the above-mentioned work of Rudolf Schmidt[67]. Despite the greatest care, a correct diagnosis was not made. It is worthy of note that in the first of these two patients attacks of painful colic, inaugurated by distressing intestinal contraction and very loud borborygmi, were present. The diagnosis lay between intestinal sarcoma and cancer. The autopsy showed a stenosis, which, however, was not produced by the neoplasm, but by adhesions of two sarcomatous degenerated coils of intestine. In the second case, complicated by tuberculosis of the pulmonary apices, the clinical picture was that of peritoneal tuberculosis. Œdema was present in both instances.

Naturally the presence of a tumour is indispensable for the diagnosis; a tumour in the second case might have made diagnosis possible. The above coincidence of pulmonary tuberculosis and lymphosarcoma shows the importance of a careful history. When a characteristic tumour is present, the diagnosis of sarcoma of the small intestine presents no insurmountable difficulties, particularly when the age, rapid development, and absence of stenotic symptoms are taken into consideration.* When a well-defined tumour is absent a correct diagnosis is only accidental, although the course of the disease may justify the diagnosis of a malignant affection.

When tuberculosis is present or suspected, particularly when a serous effusion is present, the diagnosis will often lie between peritoneal tuberculosis and an obscure abdominal tumour (cancer, sarcoma, lymphosarcoma). The coincidence of tuberculosis and lymphosarcoma has already been mentioned.

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* [Even in a young person presenting a tumour the diagnosis is by no means always possible. Two of Libman's cases (loc. cit.) very closely simulated appendicitis. In the one, M. G., age 18 years, there was a history of only one day's standing, with such acute symptoms that the case was thought to be a perforated appendicitis. In a third case the diagnosis lay between sarcoma of the kidney, tubercular peritonitis, and sarcoma of the peritoneum, with *possible* primary tumour of the intestine. It must be remembered, too, that cancer of the intestine is by no means rarely met with in young individuals (see p. 297).—TR.]

TREATMENT

The treatment of intestinal sarcoma and lymphosarcoma is not different from that of cancer; we therefore refer to the section on the therapy of intestinal cancer. Occasionally special symptoms (e. g., diarrhœa) require treatment other than that given under cancer.

As shown by the observations of Michel[68], Gilford[69], Hahn[40], Engström[70], and others, surgical treatment may at times be successful, even though the sarcoma be situated in the upper segments of the intestinal canal. Favourable results are, however, much more frequent in sarcoma of the rectum. Recurrences generally occur sooner and more extensively than in intestinal cancer.

Where operation is contraindicated, the systematic subcutaneous injections of arsenic, according to the method of von Ziemssen[71], have given favourable results both upon the symptoms and the general condition of the patient.

B. BENIGN NEOPLASMS OF THE INTESTINAL CANAL

Benign tumours of the intestine are extremely rare. Some possess no clinical interest, since they produce no symptoms during life. Others, however, are of greater importance from a diagnostic, therapeutic, and especially from a prognostic standpoint. It thus becomes necessary to describe these tumours.

Benign intestinal neoplasms include adenoma, lipoma, fibroma, myoma, and myxoma. In some cases we have mixed tumours; sometimes there are combinations of malignant and benign tumours (myosarcoma, fibro-sarcoma, myxosarcoma, adeno-carcinoma, etc.).

1. Adenomata and Polypi

The adenomata generally originate in the glands of Lieberkühn and have a glandular structure. They occur on the mucous membrane either as sessile or pedunculated growths (polypi); they may be either isolated or multiple; their size varies from that of a pea to a fist. They are found in all parts of the intestinal canal, but their favourite seat is the rectum. They occur at different ages, but the first years of life contribute by far the largest number of examples. In some cases these polypi are the starting points for the development of malignant growths (sarcoma and cancer). Their transition into tuberculosis has been described by Prochownick[72].

Multiple polypi of the rectum and large intestine present a particularly important type of adenomata. According to the observations of Luschka[73], Whitehead[74], Hauser[75], Schwab[76], Port[77], Holtmann[78], and others, they form tumours of various sizes which sometimes extend along the entire length of the large intestine from the rectum to the ileo-cæcal valve. Hauser and Port have shown that they may even extend into the small intestine and the pyloric orifice of the stomach. The interest in these cases lies particularly in the severe bleedings which they cause, and in their tendency to carcinomatous degeneration (Helferich—Port[77], Bardenheuer[79], Smith[80], Handford[81], Paget[82], Hutchinson[83], Makins[82], Hauser[75], Holtmann[78]). A very striking fact is a certain hereditary predisposition, which was observed in no less than 4 of the 13 cases gathered by Port. In view of the relatively small number of these cases, I deem it proper to report one which I had under observation several years ago.

Polyposis recti et coli; partial extirpation of the polypoid masses; death from peritonitis.

Mrs. P. G., age 36, working woman. As a girl the patient suffered from malaria, pneumonia, and pleurisy. Has had five children; severe metrorrhagia with the last. The beginning of the present illness dates back seven or eight years. She then complained of marked tenesmus, and even then the stools were always bloody. These symptoms ceased for months, during which time she felt well. The periods of remission became shorter from year to year, and latterly her symptoms have remained constant.

Patient has about 20 evacuations a day, which are passed as follows: First, "bloody water" with mucus is passed; then normal, thin, or semisolid fæces; finally, often a large quantity of blood mixed with mucus. At times there are absolutely no fæces in the evacuations. For several years, at the end of each evacuation a nodule was protruded from the anus; this finally fell off; it looked "as if composed of small growths." After this patient felt better for a few days, except for the tenesmus and the hemorrhages. The general condition of the patient is only very slightly disturbed.

Status Præsens.—Anæmic woman; normal circulatory and respiratory organs; ptosis and atony of the stomach; displaced right kidney; no other abdominal irregularity on palpation; intestines not sensitive to pressure. On rectal examination there were found grapelike masses as large as peas or beans, which consisted of broad-based excrescences ; about 20 of these were easily removed by the examining finger. After rectal lavage, blood and several pea-sized polypi were found in the wash water. Microscopical examination shows distinct adenomatous structure. Upon severe straining a deep-red tumour, about the size of an ostrich egg, protruded itself. It consisted of numerous large and small polypi, and of several hemorrhoidal nodules.

Since extirpation (see Fig. 30) of the rectum seemed impossible, the mass was partially removed on July 25, 1893. During the operation the peritoneum was opened. Death from peritonitis three days later. Autopsy showed that the entire length of the large intestine was carpeted, as it were, with innumerable large and small polypi.

Adenomata, particularly of the rectum, are easily diagnosticated, especially, when the protrusion of one or two polypi, as so frequently happens, allows of a direct macroscopic and microscopic examination.

Of the *subjective* symptoms hemorrhage and tenesmus particularly necessitate a digital examination. If the patient strains during such examination, the finger may draw down a single or more often multiple polypi out of the anus; these may then be carefully tied and cut off and their structure immediately studied.

Even when the tumours are situated higher up, their recognition is not very difficult; the soft consistency, the well-defined limits, the pedicle, the absence of ulcerations, particularly the sharp localization of the process, are unmistakable. However, the fact that malignant processes may develop upon the bases of such polypoid growths, of which I myself have observed two instances, must warn the physician to be guarded in the prognosis. If multiple rectal polypi are present we must suspect further extension of the growths into the intestinal canal.

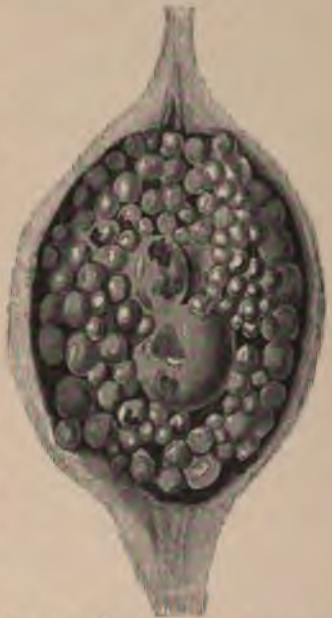

FIG. 30.—MULTIPLE POLYPI OF THE RECTUM. (Personal observation.)

II. Lipoma, Myoma

Lipoma is more frequent than myoma. There are scarcely two dozen clinically observed cases of myoma, but since the advance of surgery the cases of intestinal myoma reported seem to have materially increased. The remaining benign intestinal tumours (angioma, myxoma, teratoma, and others) possess as yet no clinical significance.

Lipomata generally originate in the submucosa, their most frequent seat being the large intestine and rectum. Their size varies considerably; generally, however, they are of an appreciable size (up to that of a child's head). They occur either isolated or multiple. The diagnosis of lipoma is only possible when they are situated in the rectum and produce symptoms. In other cases where no tumour is felt either by vaginal or abdominal examination, a definite diagnosis is impossible. Occasionally expulsion of the lipoma (Castelain[83], Albrecht[84], Link[85], Paci[86], etc.) has revealed the cause of the symptoms.

Myomata of the intestinal canal arise partly from the mucosa and submucosa, partly from the subserosa. Following Virchow, those from the mucosa and submucosa are known as internal, and those from the subserosa as external myomata. Steiner[87], who has made an exhaustive analysis of benign neoplasms of the gastro-intestinal canal, reported 19 cases of internal and 15 of external myomata. The tumour is usually situated in the small intestine, and only exceptionally in the large intestine (inclusive of the rectum). The duodenum is very rarely affected. Myomata vary very much in size; they may be as small as cherries or as large as a man's head. As regards the age at which myomata may occur, they are by no means limited to young individuals; cases have been reported in persons forty, fifty, and even eighty years of age.

Only when there is a definite tumour can the *symptomatology and diagnosis* of myomata be considered. Such palpable tumours are found in but a limited number of cases, but even then the diagnosis will be made in very exceptional instances. The diagnosis will be possible in internal myomata, when certain complemental symptoms or complications which tend to clear up the clinical picture are present. Among these we might mention the development of a more or less complete invagination, which, according to Steiner, was observed no less than 7 times in 18 cases. In a case of this nature described by Fleiner[88], in view of the chronic course of the disease, of the variation of the patient's condition from good to bad, etc., the diagnosis of myoma was made with a fair degree of probability. Besides an invagination, the gradual development of an intestinal stenosis or an obstruction is of great diagnostic significance; in such cases, days, weeks, months, or even years of absolutely good health may intervene.

The *symptoms of external myomata* are only rarely sharply defined. Evidences of stenosis may be present, thus making the connection between the neoplasm and the intestinal canal more probable. Complete obstruction by kinking, incarcerations, or volvulus, appears to be still more infrequent. In several instances the intestinal contents were bloody, and contained much mucus. Death from profuse hemorrhages from the bowel has also been described.

According to Steiner[87] the following data indicate external myomata: The presence of a slowly growing, intraperitoneal (sometimes also retroperitoneal) tumour, hard and nodular on its surface, and having no connection with the genital organs; passive movements of this tumour cause dragging pain in the abdomen; there may be symptoms of an obstruction of the intestinal lumen, and finally intestinal hemorrhages.

Despite these data we shall hardly be able to diagnosticate other than the presence of an intestinal neoplasm of unknown nature.

Myomata of the *rectum* require a brief mention. According to Steiner, only 6 cases have been observed. Here also we distinguish between the external and internal myomata. The symptomatology of the former is quite similar to that of pedunculated polypi—passing of blood and mucus, tenesmus, and, when of large size, evidences of rectal stricture. The diagnosis of myoma recti may be made when by rectal (and vaginal) examination a rather smooth, movable tumour, with a more or less thick pedicle attached to the mucous membrane is felt.

The clinical picture of *external* rectal myomata is much less characteristic. When they reach considerable size they compress the pelvic organs and become adherent to the latter. The only remaining diagnostic data are the occurrence of rectal hemorrhages and the obstruction to evacuations. It would also be of diagnostic importance to demonstrate that the tumour is not connected with the genital organs.

TREATMENT

No matter in what portion of the intestinal canal the benign tumour may be found, internal treatment can be of little benefit. We shall have to limit ourselves to the treatment of the symptoms—pain, stenosis, enterorrhagia, etc.

Expectant treatment may be successful, for benign tumours, especially when situated in the lower intestinal segments, are sometimes spontaneously expelled. As already mentioned, this has been observed in lipomata, adenomata (polypi), and myomata (Pellizari[89], Heurteux[90]).

Regarding simple pedunculated polypi of the rectum, their removal is easily accomplished with the scissors or the galvano-cautery. The case is quite different, however, with larger myomata of the intestinal canal. In many cases we shall have to be content with palliative treatment. Should dangerous symptoms occur (invagination, complete or beginning intestinal obstruction, severe, repeated intestinal hemorrhages), operative extirpation of the tumour is indicated. Successful operative results in internal myomata have been reported by Fleiner, Czerny, Lockwood, Albert, Rosi, Fenger, and Holländer[91]. External myomata have also been successfully operated on (Wölfler, Babes-Nanu, Kukula, and Krukenberg).

Internal rectal myomata offer no great difficulties to surgical treatment, for ligation of the pedicle and removal of the tumour is usually sufficient. The treatment of external myomata of the rectum is the same as that of rectal cancer. Removal of the tumour by laparotomy is indicated when its growth extends beyond the small pelvis. Cure resulted in 3 out of 4 cases of external rectal myoma (Berg, Senn, Pfannenstiel[92]).

General intestinal polyposis is not favourable either for internal or surgical treatment. Our therapeutic aim will be limited to decreasing the hemorrhages by appropriate internal medication (ergotin, hydrastin, witch-hazel), or by astringent enemata (ferripyrin, tannin, aceto-tartrate of aluminum). Partial extirpation of the growth may be symptomatically advantageous. Thus, in a case of polyposis of the jejunum and ileum recently reported by von Karajan[93], complete cure resulted from extirpation of 10 of the largest polypi. In a case reported by Sklifassowski[94], the formation of an artificial anus seemed to have been followed by some benefit.

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CHAPTER XVIII

INTESTINAL STENOSIS AND INTESTINAL OBSTRUCTION (ILEUS)

A. INTESTINAL STENOSIS

Preliminary Remarks.—Of all disturbances of the intestinal canal, the most serious are those which interfere with the normal passage of the fæces. Where they do not directly threaten life, such disturbances cause a number of extremely distressing symptoms, which, unless relieved, gradually undermine the constitution of the patient and finally produce death. At first, the variety and situation of the obstruction seem of secondary importance to the dangers arising therefrom; for, with the single exception of fæcal impaction, the danger in all varieties of intestinal obstruction is practically the same. It would thus seem that the trouble taken in the study and classification of the various forms of intestinal obstruction has but little practical value. A more detailed knowledge and more careful study will show that this idea is incorrect. The clinical features of intestinal obstruction are never the same, and the recognition of the differences in different cases is important for diagnostic, prognostic, and therapeutic purposes, particularly in surgery. There are only a few, though perhaps very important, varieties of intestinal obstruction which really offer insurmountable diagnostic difficulties.

In every case of intestinal obstruction the following facts must be determined before a diagnosis can be arrived at:

1. First and most important, the establishment of the presence of intestinal obstruction or stenosis.
2. Determination of its situation.
3. Determination of its anatomical causes.

In order to understand the symptoms of individual cases, and in view of the practical purpose of this work, it appears to me preferable to first give the *general symptomatology* of intestinal strictures and occlusions, and then the symptomatology and diagnosis of the several kinds of obstruction. The differential diagnosis will be treated of in a separate section, and, finally, the treatment of all forms will be discussed together. Owing to their extreme rarity and because of their lack of practical clinical significance, congenital stenosis and occlusion will be left entirely out of consideration. Stenoses of the rectum are described in the chapter on Diseases of the Rectum.

GENERAL SYMPTOMATOLOGY OF STRICTURE OF THE INTESTINE

The idea embodied in the term "intestinal stricture" already seems to imply a gradual development with well-defined symptoms only in advanced stages of the disease. Patients, as a rule, complain of the characteristic symptoms of stricture only when the process is relatively far advanced. There are, however, important exceptions to the usual course. In the first place, a chronic stricture may develop shortly after a complete obstruction (pseudo-ligaments, hernial orifices, partial obstruction by gallstones, foreign bodies, intestinal concretions, intussusception, compression) (Leichtenstern[1]). On the other hand, the characteristic picture of chronic intestinal stenosis may for a time be present; then, either through muscular paralysis, marked increase of the stricture, or through impaction of foreign bodies (generally undigested food), the chronic stenosis suddenly changes into a complete intestinal obstruction. In other cases, again, the change is less acute. For example, a patient has passed through several attacks of intestinal stenosis, each one more severe than the preceding one, so that it is probable that the next attack will be one of complete obstruction.

The clinical picture varies in accordance with the site and degree of the stenosis.

Regarding the site, we may, in general, differentiate between strictures of the upper and of the lower segments of the intestines, the former including the portions from below the pylorus to the jejunum inclusive, the latter those from the ileum to the rectum. Both have this in common, that the normal onward movement of the bowel contents is either delayed or entirely interfered with. As a natural result a dilatation gradually develops above the stenosis, and acts as a reservoir for the retained fluid or solid masses. It is quite evident that this will occur to a lesser degree in stenoses above the jejunum than in those lower down. It is clear that the latter variety demands considerably greater expulsive power of the intestinal muscles than does the former. Finally, in deep-seated stenoses the recoil contraction can have little effect in causing a backward movement of the solid matters in the intestine; in stenoses of the upper bowel, however, where the contents are fluid, very little force is required for their regurgitation.

From these differences the chief symptomatological distinctions between the two types arise.

Let us begin with the *small intestine.* In general the symptoms point rather to disturbances of gastric than of intestinal functions. The subjective symptoms are those of chronic overdistention of the stomach from the backward pressure of the retained duodenal and jejunal contents—viz., fulness, pressure, pains, eructation, nausea, vomiting.

Naturally this permanent stasis is not without its effect upon the appetite and nutrition—both of these suffer more or less. The increasing vomiting will also affect or retard the evacuations, but not nearly as much as in stenosis of the lower intestinal segments. In general, meteorism is very moderate and limited to the epigastrium. In marked stenosis the fluid chyme can pass downward through the intestinal canal, or find its way upward to the stomach; therefore in stenoses of the upper part of the intestines, intestinal peristalsis, or intestinal rigidity is rarely seen. For the same reason severe paroxysms of pain are also absent.

The clinical picture of deeply situated intestinal stenosis, particularly that of the *large intestine*, is quite different. Here the evidences of muscular insufficiency become very prominent. In the first place there is constipation, which may be the only symptom in the beginning, or throughout the disease. But constipation in itself allows us to draw no conclusion respecting the ominous changes occurring within the intestinal lumen. Even this symptom is sometimes absent. Nothnagel[2] cites a case of cancer of the sigmoid flexure in which natural firm evacuations occurred one day before complete intestinal occlusion set in; and every experienced physician can cite similar instances. Although constipation is one of the most frequent symptoms of stenosis of the large intestine, we may, as in intestinal cancer (page 311), occasionally have the reverse condition, namely, diarrhœa. The latter is caused either by decomposition of the dejecta above the stricture, or by irritation of intestinal ulcers (stercoraceous ulcers, distention ulcers (Kocher)). Finally, constipation may alternate with diarrhœa.

In connection with obstinate constipation, spasmodic pains may sooner or later develop. These pains are distinguished from those of ordinary flatulent colic by their frequent recurrence and increased duration and intensity. At a late stage they may be continuous.

In contrast to flatulent colic, in which the whole or greater part of the abdomen is tympanitically distended, the meteorism even in advanced stenosis of the large intestine is generally inconsiderable. Only after complete obstruction with intestinal paralysis, do general meteorism and simultaneous cessation of intestinal contractions occur. When we consider that the gases developed by the accumulated intestinal contents are powerful stimuli to peristalsis, it is not difficult to understand why meteorism is absent as long as the functional activity of the bowel is preserved.

The most important accompanying symptom of painful intestinal contraction is the occurrence of *visible, spasmodic intestinal peristalsis*, a phenomenon which Nothnagel[2] appropriately called "intestinal rigidity." These severe visible and palpable spasmodic contractions of the intestines in their effort to force their contents through the stricture, are analogous to the attempt of the uterus to force the child's head through the relatively small outlet by increased muscular action. This intestinal contraction is a favourable symptom in so far as it proves that the hypertrophied intestinal muscle still possesses a certain amount of power; on the other hand, it demonstrates that the stenosis is so great that extraordinary efforts are necessary in order to pass the obstruction.

In the General Division (page 69) we have already discussed the phenomena of intestinal rigidity. We shall return to its significance and varieties in the section on diagnosis.

Formerly great value was laid upon the *consistency of the stools*. It was believed that pointed, narrow-calibred stools were characteristic of stenosis of the large intestine. This error has found its way even among the laity, and every physician can cite cases of imaginary stenosis in which the patients complain of constipation and habitually examine their own stools. Laparotomy has been performed in a number of these patients.

We now know that in intestinal stenosis the stools have no characteristic appearance, for we find similar stools in spasmodic constipation, intestinal atrophy, in membranous enteritis, and even in ordinary intestinal catarrh. It appears to me that the idea of so-called stenotic dejections has originated in great part from strictures of the lower large intestine (from the sigmoid downward), for in that part of the tract characteristic stools do actually occur. Under certain conditions, blood, pus, and mucus may be mingled with the evacuations. These anomalies, however, are not peculiar to the clinical picture of intestinal stenosis as such, but depend upon the underlying intestinal disease.

SPECIAL SYMPTOMS AND DIAGNOSIS OF INTESTINAL STENOSIS

In well-marked cases the diagnosis of intestinal stenosis is easy; in other cases only a probable diagnosis can be made; in some the diagnosis is impossible. This depends partly upon the site of the occlusion and partly upon the prominence of the symptoms, which vary in the different kinds of stenosis.

(*a*) *Stenosis of the Small Intestine*

Analogous to the classification of malignant disease of the small intestine, benign stenoses are also divided into the suprapapillary and infrapapillary, jejunal, and ileal forms. As already stated in the chapter on Intestinal Cancer (page 303), the diagnosis of suprapapillary stenosis is rarely possible. Its clinical picture is so similar to that of pyloric stricture, that, despite the most careful examination, a correct diagnosis is the exception and a false diagnosis the rule. The diagnosis can be made with a fair degree of probability only when the subjective symptoms point to the duodenum as the certain site of the lesion. In the following case the diagnosis was made with the greatest possible clinical certainty before operation.

Stenosis of the superior portion of the duodenum following an incarcerated gallstone. Tetany. Gastro-enterostomy. Death.

Mrs. N., fifty years old, has been suffering from attacks of gastric colic for over twenty years. These would often cease for years, and later return accompanied by very intense pain. The attacks lasted minutes to hours, and were frequently accompanied by chills, fever, cold sweats, and vomiting. Twenty years ago had jaundice and clay-coloured stools. She cannot remember whether this occurred in an attack of colic or not.

Patient has been free from attacks for the last eight years, and felt well till Christmas, 1897. She then began to have a feeling of discomfort in the epigastrium, and foul-smelling eructations, generally toward evening; of late there have been acid burning eructations early in the morning, and regurgitation of stomach contents. Since the end of January there has also been vomiting of large quantities of fluid, foul-smelling masses, but not of blood. Patient often has the feeling as if the stomach "works strongly" after meals. Always relieved after vomiting. Marked loss of weight; appetite good; constipation obstinate.

Status Præsens.—No evidence of cachexia; skin of yellowish colour. Slight emphysema; heart sounds normal.

Abdomen.—Abdominal walls are moderately fatty; no visible gastric or intestinal peristalsis. When the stomach is empty loud splashing and succussion sounds are present to almost a handbreadth below the umbilicus. Epigastrium not sensitive to pressure; no pathological dulness or resistance. The border of the liver indistinctly palpable; hepatic dulness diminished, and begins at mammary line at upper border of the fourth rib. Spleen and kidneys negative.

The vomitus consists of undigested food remnants, is neutral in reaction, contains large quantities of sarcinæ, yeast fungi, muscle shreds, fat, starch, stearic acid bundles. No long bacilli.

During the next few days the stomach contents were expressed during the fasting condition. Each time food remnants rich in HCl, and showing microscopically the above-mentioned substances, were obtained. The stools consisted of scybala, containing enormous numbers of fatty acid crystals. The quantity of urine was between 400 and 900 centimetres in twenty-four hours, and contained an abundance of indican. Otherwise it was normal.

Treatment.—Fluid and semisolid diet. Daily gastric lavage; nutrient enemata.

Course.—On March 17th, after complaining of nausea and a feeling of abdominal distention, the patient suddenly had a spasmodic attack limited exclusively to both hands. The fingers were flexed, but could be passively extended. Patient was collapsed; pulse could not be felt; face cyanotic; eyes staring and glassy; complete consciousness retained throughout the attack, which lasted for half an hour.

Two hours later, despite repeated examinations, neither Trousseau's nor Chvostek's phenomena could be elicited. There was no muscular irritability, no sensory disturbances. Repeated examinations always gave the same results.

On March 20th, after previous nausea and abdominal pressure, there was numbness and stiffness of the fingers.

Since the stomach contents always increased, and renewed attacks of tetany were feared, the patient was transferred to the private clinic of Professor Hahn on March 28th.

The probable diagnosis was duodenal stenosis, situated high up, consequent upon incarcerated gallstones.

Operation on March 31, 1897, by Professor Hahn. Slight attack of tetany during narcosis.

Laparotomy.—The pylorus is free; underneath it is felt a large gallstone. In order to reach the latter a horizontal incision is made in the region of the gall bladder. Gall bladder and parts about the gallstone tightly adherent to the duodenum. The gallstone partly compresses the duodenum and partly protrudes into its lumen. The adhesions are separated and the stone lifted out of its bed, during which manipulation the duodenum is torn. Intestinal suture, then gastro-enterostomy. The stone is composed of cholesterin. Collapse and death the following day.

In a second case—a bookkeeper, fifty years old—the probable diagnosis of high duodenal stricture and calculus, resulting from cholelithiasis with icterus which had lasted many years, was made. Gastro-enterostomy was performed by Dr. Hahn and the diagnosis was confirmed. The patient was cured.

These two cases are interesting because they show that the suspicion of calculous obstruction of the pylorus and duodenum may be awakened by the occurrence of attacks of cholelithiasis for many years, and by the later occurrence of dilatation of the stomach. In view of the close relation between the gall bladder and duodenum, we are justified in suspecting the duodenum as the site of the stenosis.

On the other hand, as demonstrated by a case recently described by Wegele[3], the diagnosis of a high calculous duodenal stenosis may be very difficult during life, and even during the course of operation.

The diagnosis of low (infrapapillary) duodenal stenosis may be made with a much greater degree of probability. This has been shown by the more recent observations of Leichtenstern[4], Cahn[5], Riegel[6], Hochhaus[7], Schüle[8], Reiche[9], Herz[10], Pic[11], Rewidzoff[12], and myself[13]. From the writings of the authors just mentioned, the following may be considered typical symptoms of infrapapillary stenosis:

The most important *subjective symptoms* are functional disturbances of the stomach, like those which occur in gastrectasis—viz., diminished appetite, feeling of pressure and fulness, or even of intense pain after taking food, eructations, nausea, vomiting, constipation, decreased diuresis, and marked loss of weight and strength. If in conjunction with these symptoms there is evidence of previous disease of the duodenum and its surroundings (enterorrhagia, duodenal ulcer, disease of the pancreas, gallstones, cholecystitis, cholangitis, and icterus), we must suspect disease of this segment of the small intestine. If, furthermore, the vomitus is constantly bile tinged, the suspicion becomes a probability, and, as regards the *site* of the stricture, a certainty.

The *objective symptoms* chiefly depend upon the disturbed stomach motility, the changes in the gastric secretion being of secondary importance. The stomach may be of normal size or dilated (cases of Riegel, Schüle, Herz, and others).

Slight visible peristalsis in the neighbourhood of the pylorus may be present, but is generally absent (Schüle). *Meteorism* is only moderate or may likewise be entirely absent. When present, it rapidly disappears through the eructations and the vomiting (Leichtenstern[4], Herz).

The constant presence of bile in the stomach is the most important evidence of infrapapillary stenosis. This is best observed in the morning after the stomach had been washed out the previous evening. With the bile the duodenal secretion (*succus entericus* and pancreatic juice) is forced into the stomach, and thus it is sometimes possible to exclude severe disease of the pancreas by the presence of active pancreatic juice (Boas). That under diseased conditions pancreatic digestion may still continue, is proved by a case recently reported by Wilms [14], of a deeply situated duodenal stenosis resulting from compression of a pancreatic cancer. Pertinent conclusions can only be drawn when the digestive tests are negative.

In these cases the secretion of gastric juice depends upon the amount of regurgitated duodenal contents, the duration, and perhaps also upon the nature of the disease. Varying amounts of HCl have been found in the different kinds of stenosis of the descending portion of the duodenum. According to Riegel's and my own experience, the same individual may at one time have abundant hydrochloric acid in his stomach, and at other times none at all. As I have demonstrated [15], the presence of bile and of pancreatic juice interferes with the digestive properties of the gastric juice only to the extent that the latter is neutralized. If the hydrochloric acid preponderates in the mixture, the gastric juice has as active digestive powers as in the normal. On the other hand, in such a mixture, even though it be made alkaline, the pancreatic ferments will be destroyed (probably due to the acid).

Microscopic examination may show evidences of gastric fermentation (yeast, sarcinæ, bacteria of various kinds, etc.); it may also show the long bacilli generally found in lactic acid fermentation.

Discolouration of the stools and increase of indican in the urine (Boas) are of diagnostic significance. Both of these changes may, however, be absent. According to Herz, bismuth administered by the mouth does not reappear in the stool. This can only be the case in very marked stenosis of the lower portion of the large intestine. In connection with other symptoms, it might possess diagnostic value.

If palpable changes are not present (tumour of the duodenum or of its surrounding tissue, adhesions, etc.), the diagnosis of the underlying cause of the stenosis is very difficult. The age and sex of the patient, and, as already mentioned, the clinical history, are of value. In women in whom there is a history of frequent attacks of stomach ache, or of icterus, we must suspect impacted stones, which have either ulcerated through the bile duct and produced peritoneal adhesions with the duodenum and compression of the latter, or small concretions which have passed through the common duct into the lower portion of the duodenum, and produced a spastic obstruction of the latter (cases of Hochhaus, Schüle, Herz, and others).

In men in the prime of life we must think of duodenal ulcerations as the cause of stenosis, especially if the characteristic symptoms of this disease have been present. Several years ago I observed and reported two cases of ulcer of the duodenum with resulting stenosis of the descending portion. In one case, which I shall now describe, the autopsy confirmed the diagnosis.

Deeply situated duodenal stenosis following cicatricial contraction from duodenal ulcer. Gastro-enterostomy. Death.

Patient, a shoemaker, says that since early childhood he has suffered from intestinal disturbances (diarrhœa, anorexia, occasional biliary vomiting), so that his development was much retarded. In his seventeenth year he had typhoid, from which he very slowly recovered. He felt better for a time, but frequently thereafter suffered from obstinate vomiting, with diarrhœa. Certain articles of food were said to be passed undigested. The patient also complained of flatulence, headaches, inability to work, and lassitude.

Examination showed a poorly nourished man with normal organs of respiration and circulation. Nothing special found in the nervous system. The abdomen is somewhat tympanitic, but no new growth can be made out; liver and spleen normal; kidneys cannot be felt. In the right hypochondrium, corresponding to a prolongation of the parasternal line, there is a resistance, which is sensitive, particularly upon deep pressure. The limits of the stomach are normal. After large meals slight splashing can be made out in the epigastrium. Distention of the stomach with air plainly shows the larger curvature at the level of the umbilicus. The largest diameter obtained by extreme distention is 13 centimetres. Rectal examination and insufflation yield nothing special.

The examination of the stomach contents, made more than 100 times in three years, shows the permanent presence of bile and the absence of food remnants during fasting. The reaction of the stomach contents was at first alkaline; after a test breakfast they were occasionally slightly acid. The gastric contents, both in the fasting condition and after eating, always possessed peptic power—i. e., pancreatic juice was mixed with the bile. Later the picture changed; the gastric contents became acid and gave a decided hydrochloric acid reaction, though the contents were still bile-tinged. After some time the first-mentioned condition was again present. There was again distinct HCl reaction.

The patient's condition varied. Diarrhœa was present. At times the patient complained constantly, however, of a painful pressure along the right parasternal line, lack of appetite, headaches, fatigue, etc.

Since these symptoms recurred during the following months, the patient consented to a gastro-enterostomy; this was performed in January, 1892. At the laparotomy the pylorus was found extremely dilated, so that it was very difficult to determine which was the duodenum and which the pylorus. Externally, besides a few adhesions of the duodenum to the liver, nothing noteworthy was discovered. The patient died the following day.

Autopsy showed the condition of the stomach and duodenum pictured below (see Fig. 31). The pyloric ring was extraordinarily dilated, and toward the upper portion of the duodenum almost obliterated. The first portion of the duodenum was also extremely dilated and showed several ragged, eroded ulcers. The descending portion also contained several similar small ulcerations, somewhat larger than lentils, with numerous cicatricial strands. The latter had considerably narrowed the lumen of the descending portion.

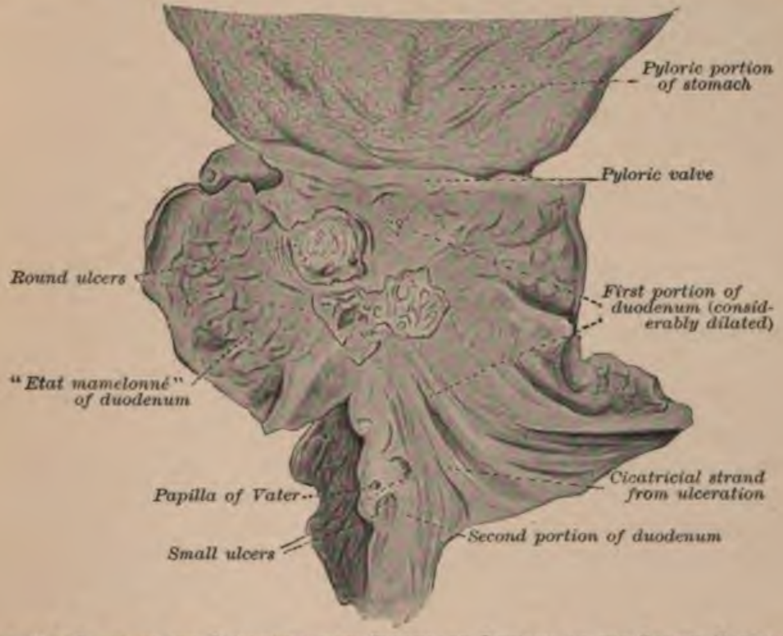
Pyloric portion of stomach
Pyloric valve
Round ulcers
First portion of duodenum (considerably dilated)
"Etat mamelonné" of duodenum
Cicatricial strand from ulceration
Papilla of Vater
Second portion of duodenum
Small ulcers

Fig. 31.—Ulcer of the Duodenum, with Secondary Stenosis of the Second Portion and Dilatation of the First Portion. (Personal observation.)

Where tubercular symptoms are present we must consider the possibility of tubercular ulcerations, though, as shown by a case of Herz,[10] under these circumstances other factors may produce the stenosis (e. g., in the case just mentioned, a peritoneal strand).

In advanced age we must first think of cancer of the duodenum and its surrounding parts as the cause of the stenosis. This question is discussed in the chapter on Intestinal Neoplasms, to which, in order to avoid repetition, we refer.

The above list by no means includes all the engendering causes of duodenal stenosis. To describe them all would be of no value, because the diagnosis is scarcely ever possible during life. It is sufficient to simply mention the other possibilities, so that they may be taken into account in appropriate cases. These are lymphomata, sarcomata, kinking, compression by metastatic tumours, pancreatic cysts, fat necrosis of the pancreas, cancer of the pancreas, retroperitoneal tumours, etc.

Stenosis of the Jejunum and Ileum

Isolated stenoses of the jejunum and ileum are rare. The majority owe their origin to the adhesions and kinking produced by inflammatory adhesions with the (female) genitals, the appendix, inflamed and reduced hernia, etc.

In rare instances healed or partially healed tubercular ulcerations are the cause of the stenosis. Fibrous stenosis, analogous to hypertrophic pylorus stenosis, is of anatomical interest only. It has been studied particularly by French and English writers ("*enterite sclereuse*," "plastic linitis," "*cirrhosis intestinalis*"), and affects partly the stomach and partly the liver, peritoneum, and isolated portions of the intestine. Cases of stricture of the small intestine from unknown cause (syphilis enteritis) have been reported; only a very few are of carcinomatous nature (Petrina, Chouquet, Letulbe, Brosch, E. Hahn, Reinke, Wernich, Küttner[16]). Tubercular stenoses may occur isolated, but (more frequently) are multiple. In a case described by E. Fränkel[17], and another by Hofmeister[18], 12 strictures were found. The strictures are usually situated in the ileum, occasionally in the cæcum, and but rarely above or below these parts.

On the whole, the clinical picture of jejunal and ileal stenosis is but little characteristic. The higher the stricture in the jejunum the more will the symptoms of disturbed gastric digestion (particularly vomiting) preponderate; and the greater the degree of the stenosis the more apt is the vomiting to be fæcal or feculent in character. The nearer the stricture is to the cæcum the more marked are the actual intestinal symptoms—constipation, or alternating constipation and diarrhœa, meteorism, severe colic, visible and palpable intestinal contraction, particularly in the middle of the abdomen—symptoms which differ little from those of stenosis of the large intestine.

As shown by the observations of Litten[19] and E. Fränkel[17], marked tubercular stenoses of the ileum may run their course entirely, or almost entirely, without symptoms. If increasing debility does not cause death, the condition becomes recognisable by the development either of intestinal obstruction or of an acute perforative peritonitis.

In a case of multiple strictures of the small intestine, of unknown character, reported by Faber[20], besides uncharacteristic intestinal disturbances, there were marked symptoms of a severe pernicious anæmia from which the patient died. Faber explained the pernicious anæmia by absorption of certain toxic products which developed above the stenosis. The correctness of the explanation may be questioned. At all events, the occurrence of progressive anæmia in connection with stricture of the small intestine is interesting. According to Faber the same observation had already been made by Johnson and Wallis[21].

Since stenosis of the lower small intestine produces symptoms only when very far advanced, the diagnosis is very difficult. If the diagnosis of a jejuno-ileal stenosis has already been established by a careful analysis of the history and of other data, it will not be difficult to determine its character. Moreover, as above seen, we must always think of the possibility of multiple intestinal stenoses, the clinical diagnosis of which, as far as I know, has never been made.

Differential Diagnosis

The differential diagnosis must first establish the fact of a stenosis of the upper part of the intestines, and then more exactly define the situation as well as the cause of the same.

The clinical picture of high duodenal stenosis is so similar to that of pyloric stenosis, that, as previously mentioned (page 346), only especially favourable circumstances can make differentiation possible. On the other hand, so far as known, the permanent presence of bile in the stomach always points to the existence of infrapapillary duodenal stenosis. It is questionable whether the absence of bile from the stomach excludes the latter disease. In his oft-quoted work Herz concludes that in dilatation of the stomach the presence of bile may not be recognised, so that the symptoms of pyloric stenosis predominate in the clinical picture. But it requires further confirmatory evidence to determine whether this difficulty can be overcome by washing out the stomach in the evenings and examining the contents of the fasting stomach, a procedure which I always recommend.

The above data apply to the differentiation between stenoses of the upper segments of the small intestine and those of the lower. There is generally great difficulty in distinguishing between a deep stricture of the small intestine and stricture of the large intestine. *Subjectively* the occurrence of frequent diarrhœa and the gastric disturbances (nausea, vomiting, anorexia, etc.) may be of diagnostic value, but constipation, and alternate constipation and diarrhœa, may also occur in stenosis of the large intestine. *Objectively*, marked, visible peristalsis may lead to a probable diagnosis of the obstruction. We will discuss this more fully in the section on stenosis of the large intestine. The lower the stenosis the more feculent will be the masses which are regurgitated into the stomach. Finally, in deep stenosis of the small intestine, particularly when far advanced, more or less meteorism develops.

In the determination of the cause of the stenosis we must first decide whether the process is malignant or benign. If malignancy can be excluded, we should look for an etiological connection between some previous disease and the benign stricture. In stenosis of the upper portion of the duodenum we ought examine for disease of the neighbouring organs—of the liver, gall bladder, pancreas, and right kidney. Lower down we must think of adhesions, of kinking, of compression—conditions produced by disease of the female pelvic organs, the appendix, and by other local peritonitic processes. The other varieties of stenosis mentioned above are of secondary importance; although the clinical symptoms be very complete their diagnosis is sometimes impossible.

(*b*) *Stenosis of the Large Intestine*

The subjective symptoms are constipation and colic associated with nausea and vomiting. The constipation has significance only in connection with other symptoms; it may deserve consideration because of the manner of its occurrence. If a patient, particularly one in advanced life, in whom the intestinal functions have always been normal and who has not changed his diet or way of living, suddenly develop constipation, it is always a significant symptom, for habitual constipation is not a disease of advanced age, but of youth and middle age. Furthermore, its course is to be observed. In contrast to simple intestinal atony, this constipation does not develop gradually, but very rapidly, and it reaches its highest point very quickly. Laxatives become useless in a few weeks or months, and the patient is soon forced to use drastic cathartics. I have already called attention to the great practical importance of circumspection in the use of laxatives in constipation. A one-sided view of this subject should not, however, be taken, for exceptions are sometimes met with.

In other cases the constipation exists for years as a harmless complaint, before it becomes more severe. The patient who formerly got along with rhubarb must now use aloes, bitter waters, or colocynth, and these always in large doses.

In addition, spasmodic, paroxysmal intestinal pains call attention to the presence of an intestinal obstruction. In conjunction with spasmodic intestinal rigidity (soon to be more minutely described), these pains assume a significant character.

Vomiting is an important and, to my mind, not sufficiently valued symptom in stenosis. It recurs with the intestinal colic, and in itself presents nothing characteristic. It derives its importance, however, from the fact that it is extremely rare in stercoraceous flatulency. If the vomiting recurs with each severe attack of colic, it ought to warn the practitioner and cause him to suspect intestinal stenosis.

Objective Symptoms.—The most important objective symptoms are meteorism, palpable and visible intestinal contractions occurring at intervals, and changes in the character of the stools.

The *meteorism* varies according to the degree and seat of the intestinal stricture. It is scarcely appreciable in strictures of the rectum (to be described later), considerable in stricture of the descending colon, and most extensive when the stricture is in the upper segments of the large intestine. It also varies with the amount of fulness of the suprastenotic intestinal segments. It may be very moderate after abundant evacuation, and increase considerably after a few days of obstinate constipation. It depends upon the sufficiency of the hypertrophic intestinal muscle above the stricture—so much so that we should always regard increasing meteorism as a precursor of approaching intestinal paralysis (eventually in connection with peritonitis). As long as the lumen is to some extent permeable, the tympanites will be inconsiderable, and limited to one or both iliac regions ("flank meteorism," Nothnagel *), the mesogastrium, the umbilical region, or the hypogastrium. Therefore, with certain reservations we can draw important conclusions regarding the site of the obstruction from careful observation of the tympanites.

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* Loc. cit., p. 375.

We can sometimes define the extent of the tympanitic area by percussion, or better, by auscultatory percussion. Nothnagel has pointed out that in stenosis of the large intestine, instead of the normal more or less marked dulness and low resonance in the upper lumbar region posteriorly, there is often a loud and deep percussion note; this is present on both sides in stenosis of the sigmoid flexure or of the descending colon, and only on the right side in stenosis of the splenic flexure or of the transverse colon.

Visible peristalsis associated with intestinal rigidity is much more significant than tympanites. As has been already mentioned (page 68), visible peristalsis as such not infrequently occurs normally in emaciated individuals, particularly in women with diastasis of the recti muscles or ptosis of the abdominal viscera. It has also been observed as a motor neurosis, which was first described by Kussmaul under the name of "tormina ventriculi." Both forms, however, are entirely distinct from visible intestinal peristalsis, for in the latter the intestinal spasm and the circular palpable and visible intestinal contraction and rigidity are absent.

In every spasmodic peristaltic action several phases may be distinguished: a gradual onset, which is accompanied by moderate pain; steady increase of the pain up to its point of greatest severity while the bowel is contracted and rigid; finally, rapid abatement of the attack, with the occurrence of palpable and audible intestinal sounds—i. e., sounds of gas forced through the stricture.

The intestinal contraction may be limited to a small portion of the gut, or whole coils may contract, become snakelike and swollen, and again relax. The former variety (limited contraction) is observed principally in stenosis of the colon below the cæcum, the latter in stenosis of the cæcum and small intestine. The degree of the stenosis will naturally greatly influence the extent of the tetanically contracting intestines involved, and the frequency of the attacks.

Can any conclusions as to the seat of the obstruction be drawn from the configuration of the intestinal rigidity?

Based upon the experience of others and myself in this relation, this question must be answered in the affirmative. Thus it is easy to recognise a cæcal stenosis from the active peristalsis of the small intestine. Stenoses lower down are also easily recognised if rigidity of the large intestine is well defined. Nothnagel* correctly states that errors can only arise when the intestine adjoining the stricture has lost its power of contraction, and the segment immediately above acts vicariously for the latter.

The important facts regarding the character of the stools in stenosis of the large intestine have already been described in the chapter on Neoplasms (pages 310 and 311), as well as under the general symptomatology of the present chapter (page 345). In both places we have mentioned the slight significance of this symptom, and we wish to repeat here that the absence of stenotic stools does not exclude the diagnosis of stenosis of the large intestine. The admixture of pus and blood in the stools is not characteristic of intestinal stenosis, but only indicates the presence of complications—neoplasms, hemorrhoids, partial or chronic intussusception, stercoraceous ulcers, etc.

Very rarely all these symptoms are combined. In such instances we must be content with a probable diagnosis. At all events, when well-defined symptoms of stenosis of the large intestine are present the diagnosis will not long remain in doubt.

The question of the *cause* of the stenosis is a much more difficult one. The history gives us considerable assistance. It may inform us respecting previous dysentery, tubercular disease of the intestine, or of other organs, occasionally of syphilis, appendicitis, peritonitis, in women, puerperal fever and other diseases of the genital tract, incarcerated hernia, intestinal obstruction, abdominal operations. In this manner we may gain valuable hints for the diagnosis.

The examination per rectum, and in women per vaginam, is of importance and should never be neglected. Thus in an otherwise well-defined case of stricture of the large bowel, I was recently able to distinctly palpate a contracting coil of small intestine in the small pelvis. In some cases vaginal examination, or combined rectal and vaginal examination, may give important information regarding the situation and character of a suspected stenosis of the large intestine.

The external examination of the abdomen is, however, most instructive. If a tumour can be felt, and its precise nature is in doubt, a careful examination is necessary, with especial consideration of the age of the patient and the comparative frequency of the various tumours (the rarity of benign, the much greater frequency of malignant neoplasms).

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* Loc. cit., p. 376.

Differential Diagnosis

A description of the etiology has already been given. When spasmodic intestinal contractions are absent it will be difficult to avoid diagnostic errors. This will be the case, for instance, in intestinal stenosis when the obstruction is not as yet marked or can be easily overcome by contraction of the intestine above the stricture. If there is no marked disturbance of nutrition, as in intestinal cancer, the patients do not as a rule present themselves for medical examination at this early stage of the disease. As already mentioned, the presence of the initial symptoms of stricture, be they ever so slight, should make us suspect a mechanical obstruction in the bowel. If, in addition, the history confirms our suspicion and other objective signs of disturbed intestinal functions are present (diarrhœas, blood, pus, and loss of weight), it may be possible to make an early diagnosis. It need hardly be mentioned that if tumours, adhesions, and infiltrations about the large intestine are found, they offer important data for clearing up the diagnosis. Despite these favourable conditions and because of the many other etiological possibilities there will always be doubtful cases. It is impossible to consider all the differential diagnostic data, for we should become lost in the immense literature of the subject.

B. INTESTINAL OBSTRUCTION (Ileus)

Intestinal obstruction is a condition in which the lumen of one or more portions of the intestines is occluded, and the normal forward movement of the contents entirely suspended. Since the earliest days of medicine the resulting clinical picture has been called ileus (from εἰλεέω = misereor, or εἰλέω = torqueo).

In his book on intestinal diseases, Nothnagel advocates the discontinuance of the term ileus. It is true that the ileus of the older physicians, with its extremely vague meaning, has not the same significance as the ileus of to-day. The word as used at present may stand as a short and forcible designation for intestinal obstruction. For purely practical reasons we shall retain the old classification, as accepted by Leichtenstern, of mechanical, dynamic, and mechanico-dynamic ileus.

Through interference with the normal course of the fæces at any point, a number of severe symptoms soon develops. These generally begin suddenly, soon become very severe, and either cease spontaneously, or, if not cured by internal or surgical means, cause the death of the patient.

We shall describe those varieties of intestinal obstruction which are especially important to internal medicine. The obstructions produced by external hernia will be incidentally touched upon, since they are treated of in surgical text-books.

General Symptomatology and Diagnosis of Intestinal Obstruction

As a typical example, let us take an acute obstruction of the small intestines due to strangulation: An individual, previously healthy, is suddenly seized with severe colicky pains in the abdomen, vomiting of the ingesta, partially or not at all digested. There is absolute anorexia, the abdomen rapidly becomes distended, neither fæces nor flatus are passed. Intense nausea is present even when the stomach is empty. The urine is diminished, or there is anuria. There is severe thirst, particularly after repeated vomiting. The patient looks very ill as if suffering from a severe acute disease.

The vomiting consists at first of food remnants, and when the stomach is empty is bile-tinged or grayish-green; gradually it becomes feculent, and finally fæcal.

With the onset of the vomiting pain and meteorism may temporarily cease, giving new hope to the patient and relatives, or to the physician. The symptoms very soon become aggravated; the pain reappears, the abdomen becomes more tympanitic, and even by the laity the repeated severe fæcal vomiting is recognised as a sign of intestinal obstruction.

There is also a marked change in the patient's general condition. The lack of nourishment, the loss of sleep produced by the pain and vomiting, and, above all, the shock caused by the strangulation, produce a condition of deep collapse. In sharp contrast to the latter, the patient retains full consciousness, and only just before death may delirium appear. This condition of extreme collapse, which has not inaptly been compared to the algid stage of cholera, is soon followed by dissolution.

This is a short description of a typical case of acute obstruction. The several symptoms, and particularly the objective clinical picture, require a closer study. To avoid repetition it is best to analyze both together and to point out their significance. For this purpose the general symptoms relating to intestinal obstruction will first be considered, and the special diagnosis of the several varieties of obstruction will be reserved for later discussion.

The *subjective symptoms* are pain, nausea, vomiting, retention of gas and fæces.

1. *Pain.*—This constitutes the main symptom of intestinal obstruction. It is present in all forms, but it may vary in intensity and other characteristics.

The pain is most intense and continuous in obstruction of the small intestine, independent of its anatomical cause. Its severity is such that even strong people are overcome. The pains are more continuous in character than in stenosis. As I have already mentioned in the general division (page 58), Treves[22] gives great diagnostic importance to this sign. This is true, however, only with certain limitations. The seat and type of obstruction are doubtlessly prominent factors. In the first place, as several cases of incomplete intestinal obstruction reported by Treves show, the pain may be intermittent in character, and this may be the case in well-marked obstruction of the small intestines provided copious vomiting affords temporary relief to the occluded bowel segment. The continuance or discontinuance of the pain may be obscured by the use of narcotics and stomach lavage, but we must in general agree with the conclusions of Treves[22]. It is important to note whether the pain, which was continuous, shows marked remissions, for with cautious reservations this would point either to a favourable termination or to a transition from a complete to an incomplete obstruction.

The localization of the pain possesses on the whole no great significance. In the majority of cases the pain is localized in the neighbourhood of the umbilicus, but, as Treves has demonstrated, this by no means signifies that the site of the obstruction is to be sought for in that region.

We must distinguish between *abdominal sensitiveness to pressure* and subjective pain. The former is generally absent in the beginning of the disease; in the later stages it may be either circumscribed or diffuse. Circumscribed pressure sensitiveness occasionally occurs in the first days of the obstruction; it then has a certain significance, since it points either to an active inflammation of the intestine in question or to a local peritonitis. Diffuse sensitiveness, in connection with other symptoms (fever, etc.), points to a general peritonitis. Treves speaks of another form of sensitiveness which is developed late in the disease, and which is to be looked upon as the consequence of spasmodic intestinal peristalsis. In contrast to diffuse peritonitis, this sensitiveness is only moderate.

In all varieties of obstruction pain may cease toward the end of the disease. Nothnagel believes this is due to intestinal paralysis or to perforative peritonitis, but it is probably also caused by the ensuing collapse and lessened vitality.

2. *Vomiting.*—This is one of the most regular symptoms of the disease. The initial vomiting is no doubt reflex in character, analogous to the vomiting of acute peritonitis, of gallstone and kidney-stone colic, or of pregnancy. In the beginning the vomited matter almost always consists of the stomach contents or mucus, and, if severe, contains bile.

The transition to stercoraceous vomiting is recognised by the bringing up of brown, slightly fetid, or feculent masses. When the obstruction has developed very acutely, the vomiting may be fæcal from the very beginning. Fæcal vomiting consists of fluid, or occasionally of fragmentary brownish-yellow or brown-coloured masses. Vomiting of formed scybala, which is reported by well-known clinicians (Rosenstein[23], Jaccoud[24], Briquet[25]), is certainly extremely rare.

Ever since the time of Galen the cause of stercoraceous vomiting has been the subject of lively discussion. We shall briefly consider some of the theories which have been offered.

The old idea that fæcal vomiting depends upon perfect or imperfect action of the ileo-cæcal valve must be given up, in view of the fact that such vomiting occurs in obstruction of the small as well as of the large intestine. It must be admitted, however, that in the latter instance there is an insufficiency of Bauhin's valve, the valve being capable of offering only a certain relative resistance to the pressure of the stagnant fæcal mass. For a long time the existence of antiperistalsis has been disputed. That antiperistalsis may occur, has been proved by Nothnagel's experiments on rabbits with common salt, but Nothnagel has never tested his theory in intestinal stenosis. The explanation given by Haguenot in 1813, as reported by Leichtenstern[26], is ample for the comprehension of fæcal vomiting. It is briefly as follows: Strong pressure is brought to bear upon the site of obstruction by the accumulating fæces and gas, and this pressure is increased by every respiration, by every act of vomiting, and by every active intestinal contraction. Since the stagnant intestinal contents have no other avenue of escape, it is apparent that even the act of vomiting, accompanied as it is by contraction of the diaphragm and abdominal muscles, will force these masses toward the stomach. In like manner, the tympanitic intestines may act as stimuli in forcing the stagnant masses toward the stomach. Henle appropriately called this an "overflow" of fluid into the stomach. Haguenot's theory also explains why fæcal vomiting occurs much easier and more extensively in obstruction of the small than of the large intestines.

Because it is easily recognised, fæcal vomiting is one of the most important symptoms of organic intestinal obstruction. We shall see later, however, that it may also be present in simple spastic and paralytic intestinal obstruction (compare page 403).

3. *Constipation.*—In complete intestinal obstruction constipation is generally absolute, and continues as long as the disease. Neither fæces nor gas are passed. To this rule there are a very few noteworthy exceptions. Rectal irrigations may wash out small fæcal particles from the intestines below the site of the occlusion, but these are small, and always consist of fæces that were adherent to the intestinal walls (*Randkoth*). It is important to note that, in these cases, flatus is always absent. Besides such stools, Treves[22] has reported several cases with autopsies, in which more or less abundant evacuations were present during the course of the disease. Some of these cases he explained by sudden intestinal peristalsis from an intercurrent peritonitis (?). The explanation is much clearer in two of the cases. In the one instance there was an incarceration of about 20 centimetres of the ileum produced by a strand running from the transverse colon to the cæcum. An intestinal ulcer had perforated, the tension of the distended intestines was thus lessened, and the incarcerated coil was thereby enabled partly to escape from the constricting bands.

In partial volvulus of the small intestine or sigmoid flexure some fæces may be passed. Abundant evacuations are by no means rare in acute intestinal invagination.

In the article already cited, Litten[19] describes a case of multiple tubercular strictures of the small intestine, in which both profuse diarrhœa and fæcal vomiting were present. He correctly concludes that there was very advanced though not complete obstruction of the bowel.

Finally, Naunyn has pointed out the paradoxical occurrence of evacuations in obstructions by gallstones. When we come to describe the various kinds of intestinal obstruction we shall more fully analyze these different features, which are so important for the semeiology of this affection.

Objective Signs.—These are tympanites, visible peristalsis, intestinal hemorrhage, changes in the urine, and disturbances of general health.

1. *Tympanites* is always present in intestinal obstruction, but its amount varies. In acute obstruction of the small intestines, with sudden onset, it is relatively slight. The tympanites is extremely marked in obstruction of the large intestine, particularly in its most frequent form—volvulus of the sigmoid flexure. In the present state of our knowledge we may distinguish two forms of tympanites, *tympanites due to stagnation of contents* and *local tympanites.* The former develops in the large or small intestines when the lumen is obstructed by foreign bodies, invagination, impacted fæces, etc. Fluids and gas stagnate above the stenosis and distend the bowel, and the less the amount of gas absorbed the greater the distention (Zuntz and Tacker).

In the beginning of the disease, when the intestinal wall is still fairly intact, meteorism will be only moderate; it gradually increases with destruction and over-distention of the intestinal wall.

Local tympanites, already reported by Küttner[27] and Hilton Fagge[28], is very much more important than the above form. Its importance as a diagnostic factor, however, is due especially to von Wahl and his pupils, von Zöge-Manteuffel and Kader, as well as to Obalinski and Schlange. Von Wahl[29] first called attention to the apparent paradox, that in intestinal obstruction where the tympanites is most marked (volvulus, invagination, kinking) the coil in which the obstruction exists is the most tympanitic, for it is this very coil which, through disturbance of its circulation and putrefaction of its contents, is the first to be distended by gas. The formation of gas is accompanied by considerable distention of the bowel wall, and very rapid complete paralysis.

This distended, resistant, and immovable portion of the intestine is recognised by inspection from the shape of the abdomen, and by palpation, from its clearly increased resistance (von Wahl).

From extensive experiments on animals, Kader[30] has given us conclusive proof of von Wahl's theories, and has explained satisfactorily the occurrence of local meteorism. This experimenter showed that the chief cause of the meteorism lies in the disturbance of the circulation of the intestinal wall; the tympanites is explained by the increase in the size of the bowel, which is produced by the following three factors: infiltration of the intestinal wall, accumulation of fluids in the canal, and the development of gases in the interior of the intestine. The bowel segment in question soon becomes distended and tense, often within a few hours. Changes in the intestinal wall (œdema, hemorrhagic infiltration, thickening of the intestinal wall and its mesentery) occur throughout the obstructed segment and are limited to this part. Finally, there is gangrene of the intestine, frequently with perforation into the abdominal cavity. These perforations are often so minute that they are only demonstrable by distending the bowel under water. As to the diagnostic value of this fixed, distended coil of intestine, when the clinical symptoms are well marked, its presence is doubtless sufficient, and occasionally even conclusive. Its recognition necessitates a series of careful examinations. According to the description of von Zöge-Manteuffel[32], the method of the examination of this tympanitic coil is as follows: "After the history has been taken and the general condition of the patient noted, the abdomen should be carefully inspected. The smallest asymmetry must be taken into account. We should observe whether the asymmetry remains constant, whether it changes with active peristalsis, whether intestinal movements become apparent, or whether—and this is very important—there be not abnormal quiet beneath the tense, distended abdominal walls. Palpation, which follows inspection, must attempt to establish differences of resistance. On palpation, a strangulated, tympanitic coil of intestine feels quite different from the normal intestine containing fluid fæces. Requiring more space, it forces itself against the yielding abdominal wall, and is thus directly accessible to examination. If vomiting occurs, the resulting relief of tension may enable one to grasp this resistant segment. This is especially easily accomplished during chloroform narcosis."

The diagnosis of internal incarceration, strangulation, or volvulus is facilitated if such a distended immovable coil of intestine is present. Based upon von Wahl's symptom, the diagnosis of these forms of intestinal obstruction has repeatedly been correctly made, and the cases thereupon successfully operated. But von Wahl's sign is not absolutely trustworthy, when, for instance, as he himself states, a larger intestinal mass is strangulated. Schede[33] points out that the fixed intestinal coil may be covered by distended intestines lying above the obstruction. Despite all this, however, we shall have to look for von Wahl's sign in every case of intestinal obstruction.

2. *Spasmodic Intestinal Peristalsis.*—Though a classic symptom of *chronic* intestinal obstruction, spasmodic intestinal peristalsis, according to the opinion of all careful observers, is one of the rarer symptoms of *acute* intestinal obstruction. Fenwick[34] denies its occurrence; Nothnagel* mentions it, but directs attention to the great contrast between the marked intestinal rigidity in stenosis and its weak contraction in complete occlusion. Schlange[35], Obalinski[36], and Naunyn[37] consider the slight peristalsis of the "fixed coil" a very important symptom of acute intestinal obstruction. The first two authors also consider it of practical significance for it indicates vital irritability of the bowel; but this statement is disputed by von Zöge-Manteuffel. According to Schlange, peristalsis of the intestine proximate to the obstruction is best observed in strangulation of smaller coils and in obstruction by obturation. Naunyn, who has described a case in point, states that this phenomenon may also occur in volvulus without strangulation. I myself have never observed intestinal peristalsis in acute obstruction in the course of a chronic stenosis, and I consider the absence of such peristalsis as an evidence of a complete intestinal obstruction.

3. *Intestinal hemorrhages* may occur under several conditions. They are most frequent in intussusception, but, as Henoch and Wilms have shown, are occasionally found after herniotomy, after reduction of the hernia (Schnitzler), in strangulation, gallstone obstruction, and more frequently in volvulus of the sigmoid flexure and other portions of the intestines. As Tietze[38] has recently demonstrated, hematemesis may occur in intestinal obstruction as a result of severe destructive tissue changes in the proximal segment. Intestinal hemorrhage is only of significance in connection with the symptoms. When present, it speaks rather for strangulation of the small intestine than for obturation. We have already mentioned, in the chapter on Intestinal Carcinoma (page 308), that hematemesis may also occur in stenosis of the large intestine.

4. *Changes in the Urine.*—The excretion of urine is diminished on account of the very rapid collapse, the vomiting and the little nourishment taken. This oliguria is not especially characteristic, but the indicanuria which is frequently present may, according to Jaffe, have a certain diagnostic significance in relation to the site of obstruction. Numerous examinations have

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* Loc. cit., p. 211.

confirmed Jaffe's claims that marked indicanuria is present in the first days of obstruction of the small intestine, and may be of diagnostic value. If at such time marked indicanuria is absent, it would speak rather for occlusion of the large intestine. Later in the disease pronounced indicanuria may also occur in obstruction of the large intestine, and hence loses all significance. If even then no indican is present it would point still more to an obstruction of the large intestine. Indican may be increased in other affections than intestinal obstruction, and its value must always be cautiously accepted.

What is true of indicanuria is also true of Rosenbach's reaction. I have never heard of a marked Rosenbach reaction that was not accompanied by an increase of indican in the urine.

Besides indicanuria we may find albumin and casts, particularly in incarcerated herniæ (Englisch [39], Frank [40]), and in severe intestinal stenoses (von Engel [41]); hemorrhagic nephritis was observed by Israel in a case of volvulus of the sigmoid flexure [42].

5. *General Condition.*—In all varieties of intestinal obstruction the general condition of the patient suffers greatly. Naturally there are differences in the several forms of obstruction. Age, constitution, and other factors have their influence here as they do in other pathological conditions, all, however, being of secondary importance to the shock. To a certain extent the degree of the shock indicates the site and perhaps the nature of the obstruction. Thus it is a well-known fact that in occlusion of the small intestine, particularly its most frequent pernicious variety—strangulations and incarcerated herniæ—the clinical symptoms very rapidly reach their greatest severity. Obstruction of the large intestine, on the other hand, is more gradual in its development, and hence the collapse is less severe and is more slowly developed. We cannot here go into the details of the theory of collapse, in which different factors require consideration (reflex action, loss of water, intoxication, peritonitis, cardiac insufficiency, etc.).

6. *Clinical Examination.*—Clinical examination may give us valuable aid. The well-known rule—very careful examination of the external hernial orifices in cases of intestinal obstruction—should never be forgotten. We must first of all determine whether an incarcerated hernia has been actually or partially reduced *en masse.* Medical literature abounds with cases of obstruction due to incomplete or apparent reduction. Digital examination of the vagina

and rectum is quite important, and sometimes gives useful information.

In the general division we have mentioned the most important considerations regarding inspection, percussion, auscultation, and palpation. I would again mention the diagnostic value of *auscultatory percussion*, first described by Leichtenstern * and later by Curschmann [43], by means of which it is often possible to localize different portions of the intestines.

The significance of distention of the rectum with air and liquids has already been treated of at length in the general division. We again repeat that these methods may serve to confirm a diagnosis of obstruction in the lower portions of the intestine, though they are useless in obstructions higher up.

DIFFERENTIAL DIAGNOSIS OF INTESTINAL OBSTRUCTION

If the clinical picture of obstruction is well developed, and its course can be followed from the onset, the differential diagnosis between ileus and its related conditions is rarely difficult. If, however, the beginning be obscure, the symptoms not well marked, or complications present, the diagnosis becomes very difficult. Among the conditions which are generally easily differentiated from intestinal obstruction are flatulent colic, cholelithiasis and nephrolithiasis, poisoning, and cholera.

1. *Flatulent Colic.*—If observed in the earliest stages, differentiation between intestinal obstruction and flatulent colic may be difficult, particularly if the severe symptoms of obstruction have gradually developed. The history, clinical examination, and the further course of the disease are important in this connection. The history shows that the patient has often suffered from similar attacks as well as from irregular bowels. Symptoms of severe shock are absent in intestinal colic. Though flatus is generally not passed in such cases, some gas may be passed and thereby relief obtained. Vomiting, one of the early symptoms of intestinal obstruction, is very rarely present in simple flatulent colic. Further observation will scarcely ever leave any doubt as to the nature of the disease.

2. *Gallstone and Renal Colic.*—In the beginning of the attack biliary and renal colic may very much resemble intestinal obstruction, especially an obstruction situated high up. Here, again, the his-

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* Loc. cit., p. 407.

tory is important. Biliary and renal colics usually occur late in life, intestinal obstruction is found at all ages. It may be possible to palpate the gall bladder or an enlarged painful liver or painful kidney. The presence of slight icterus or of urine containing a small amount of bilirubin, or of cloudy urine containing a marked sediment, may point to the correct diagnosis. Finally, the presence of very marked indicanuria in the beginning of the disease may aid in the differentiation. With careful observation the diagnosis should soon be made.

3. *Poisoning* has several times been mistaken for obstruction. On the whole, however, error can only occur when no history can be obtained or when the disease runs a very atypical course.

4. As recent reports have shown, *cholera nostras* or *Asiatica* may cause dangerous mistakes, especially during a cholera epidemic, and particularly when that rare complication of obstruction first described by Malgaigne—obstruction with profuse diarrhœas (cholera herniaire) is present. However, careful bacteriological and other examination should nowadays make errors of this kind impossible.

5. *Peritonitis and Perityphlitis.*—Here, again, the history is the most important, occasionally the only, aid in differential diagnosis. No circumstance in the present or previous history that may have some connection with the disease should be neglected. Thus, a previous typhoid, hematemesis, dysentery, or appendicitis may clear up an otherwise obscure case. If the history renders no aid, the differentiation is very difficult in cases which are at all complicated, particularly where the onset was not sufficiently observed. Is there intestinal obstruction only, or obstruction and secondary peritonitis, or primary peritonitis, and—what is always pertinent to etiology—what is the nature and origin of the process in question?

Positive differential signs do not exist, since mild peritonitic symptoms may be present in the early stages of intestinal obstruction and thereby lead to error. By a consideration of all the important symptoms of both conditions it will be easier to arrive at a correct diagnosis. These include fever, sensitiveness to pressure, tympanites, abnormal peristalsis, vomiting, ascites, and changes in the urine.

No positive conclusions can be drawn from the presence or absence of fever. In diffuse peritonitis it may be entirely absent, whereas in intestinal obstruction the fever, when present, is apt to be only moderate. High fever from the very onset (39° C. and over) would, in itself, indicate acute, diffuse peritonitis.

The sensitiveness to pressure is sometimes of diagnostic importance, but may be absent in peritonitis and be very marked in obstruction. Still, sensitiveness to pressure is generally much more accentuated in peritonitis than in obstruction; in the latter it is severe only when peritoneal complications are already present. The situation of the area of sensitiveness is also of significance. If from the beginning it has been localized in the right iliac fossa and has remained there, the assumption of a perforative peritonitis following perityphlitis is much more probable.

In like manner the tympanites is to be cautiously employed as a differential factor. In peritonitis, as in obstruction, it may be extensive or entirely absent. When present in peritonitis it soon becomes general. This is in sharp contrast to some forms of obstruction (strangulation) in which, as we have already seen, meteorism is distinctly localized as long as there is no intestinal paralysis. But even this symptom becomes only a theoretical distinctive characteristic when we are dealing with a large sacculated peritoneal exudate which may produce the same physical signs as a distended coil of intestine in obstruction.

Well-marked, visible, and palpable intestinal peristalsis is a valuable symptom of intestinal obstruction, but unfortunately it is absent in the greater number of cases. Besides, slight evidences of peristaltic motion may easily be overlooked. The absence of all intestinal motion and sounds tends to support the diagnosis of peritonitis, but even this is no convincing proof.

Vomiting is a symptom of both diseases, and may make difficult or frustrate all differentiation. The early appearance of fæcal vomiting speaks rather for obstruction than for peritonitis. Though fæcal vomiting occasionally occurs in peritonitis with intestinal paralysis, it is by no means as frequent as in intestinal obstruction. The occurrence of a fluid exudate is found in peritonitis as well as in several forms of intestinal obstruction (volvulus, strangulation); this sign therefore possesses no differential significance.

The same is true of the amount of indican in the urine, which may also be much increased in acute peritonitis. The absence of marked indicanuria may, in connection with other signs, speak for obstruction of the large intestine.

The greater part of what has been said above also applies to perityphlitis, which is the most frequent cause of perforative peritonitis.

It is evident that those cases only ought to be differentially considered in which the clinical picture of severe intestinal obstruction is present. Aside from the history of the case, which may give us valuable information, distinct localized pain over McBurney's point, even in a case of indirectly developed diffuse peritonitis, moderate resistance, increased rigidity, or œdema of the abdominal muscles, may indicate the correct diagnosis. Where, as not infrequently happens, these data are absent the diagnosis will long be doubtful.

SYMPTOMATOLOGY AND DIAGNOSIS OF THE VARIOUS KINDS OF INTESTINAL OBSTRUCTION

I. External Intestinal Obstruction by Bands, Clefts, Fenestra, and Internal Herniæ

Of all types of obstruction the above variety is scientifically the most interesting and the most frequent, but unfortunately diagnostically the most unfruitful. There are innumerable possibilities, and it would require a monograph to carefully analyze and discuss the different varieties of this type of obstruction.

We will therefore content ourselves with a brief description of its most frequent forms. Since I have had but little experience with these various types, I shall follow the excellent description of Treves[22].

Treves distinguishes five varieties of external intestinal obstruction.

1. *Strangulation by Isolated Peritoneal Adhesions*

Since chronic local peritonitis is an extremely frequent condition, it is apparent that it is a prominent factor in the etiology of intestinal obstruction. Generally one peritoneal adhesion is present (see Fig. 32), rarely there are several. This fact is of great surgical importance, for occasionally during operation the obstruction is thought to have been overcome by ligation of a pseudo-ligament, but the continuance of the obstruction and autopsy show the presence of a second adhesion, the real cause of the constriction. The adhesions in question are circular or in the form of strands, and vary considerably in length and thickness; they vary from the size of a thread to that of a finger. According to Treves, the average length is 4 to 5 centimetres. Naturally, every adhesion which strangulates the intestine must have at least two points of attachment. One of its ends is very frequently connected with the mesentery. In other cases both ends are thus attached, and then the points of insertion are far apart (see Fig. 33, page 371). The number of possible attachments is as large as the possible adhesions of the intestines with each other and with the remaining abdominal organs. "There is scarcely any conceivable combination of connected areas which is not illustrated in the history of these adhesions" (Treves [p. 34]).

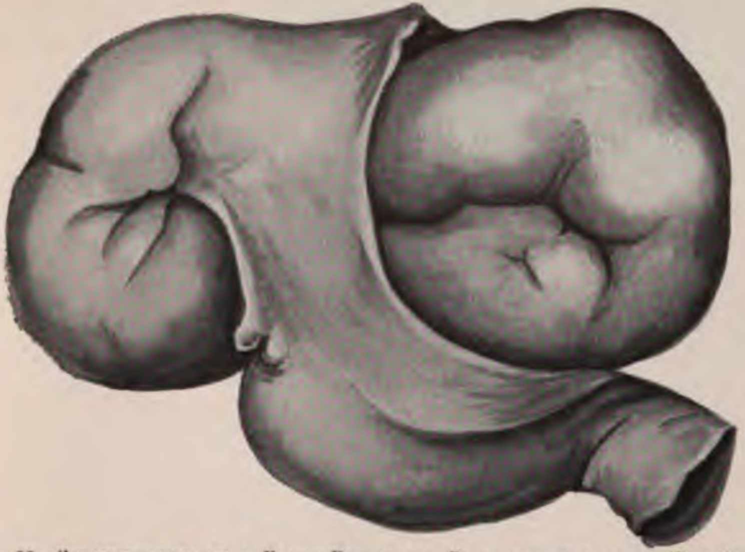

FIG. 32.—STRANGULATION BY A BROAD PERITONEAL BAND PASSING BETWEEN TWO ADJACENT COILS OF THE ILEUM. (Treves.)

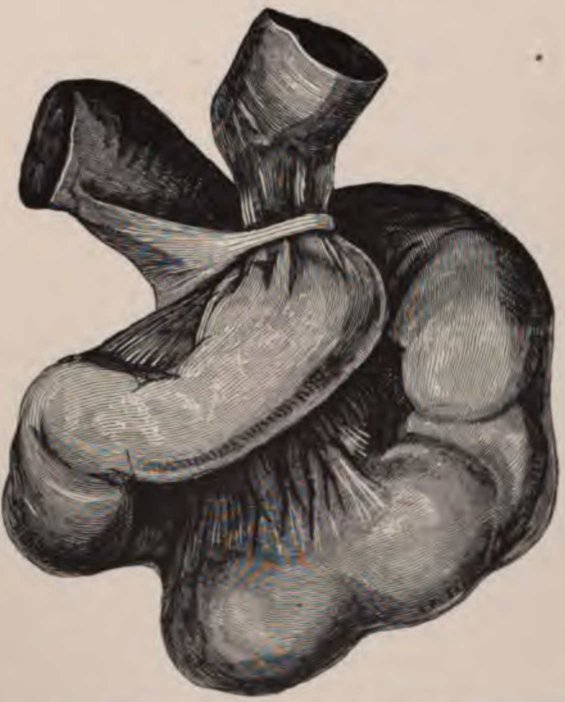

FIG. 33.—STRANGULATION OF SMALL INTESTINE BY A SOLITARY BAND ATTACHED AT EITHER END TO THE MESENTERY. (Treves.)

It is a fundamental observation that some portion of the *small intestines* is most frequently constricted, and that its most movable segment, the ileum, is generally affected.

The organs from which the bands most frequently originate are the female pelvic organs (pelvic peritonitis) and the cæcum and appendix. The number of possible variations still increases when we consider the frequent anomalous displacements of the several intestinal segments.

Besides strangulation by isolated peritoneal adhesions, there is another form in which, because of peritonitis, movable organs become fixed and thereby give rise to obstruction. To this category belong particularly the vermiform appendix, Meckel's diverticulum (see opposite page), and in rarer cases the Fallopian tubes, appendices epiploicæ, and the mesentery, which is changed into a strand. These organs form either a band or an arch under which the intestine becomes constricted, or they form a coil in which a portion of the bowel is caught. The first is the more frequent occurrence (see Fig. 34).

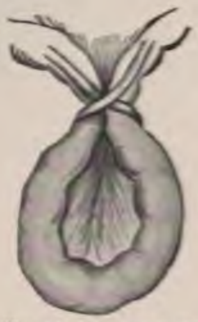

FIG. 34.—STRANGULATION OF A SMALL INTESTINAL COIL BY A LONG LIGAMENTOUS STRAND.

2. *Strangulation through Clefts and Fenestra*

This type of obstruction is rare and hence possesses only slight practical importance. Clefts and fenestra are found most frequently in the omentum and mesentery. They may be congenital, but are generally produced by trauma or by peritoneal inflammatory products, or they consist of spaces which have been formed by peritoneal adhesions between different organs (uterus, ovaries, hernial canals, appendices epiploicæ, etc.).

Discussion of the several forms and varieties is not appropriate to the scope of this work ; we therefore refer to the thorough descriptions of Treves [22] and Leichtenstern [1].

3. *Strangulation by Omental Bands*

As strands are caused by peritonitis, so bands may also develop from inflammation of the mesentery. The latter (mesenterial bands) are much larger in size than the former (see Figs. 34 and 35). They originate from traumatism, pelvic peritonitis, or, in the majority of cases, from peritonitis about a hernial sac, especially femoral hernia. The left side is more apt to be involved because the omentum generally lies in that half of the abdomen. As with peritoneal adhesions, so two or more mesenteric ligaments may be present.

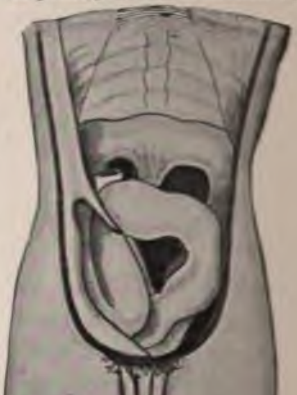

FIG. 35.—INTERNAL STRANGULATION OF AN INTESTINAL COIL BY A STRAND PASSING FROM THE OMENTUM OR TRANSVERSE COLON TO THE ANTERIOR ABDOMINAL WALL. (König.)

4. *Strangulation by Meckel's Diverticulum*

As is known, Meckel's diverticulum is due to a pervious or an incompletely obliterated omphalomesenteric duct. The abdominal end of the diverticulum is generally free; only rarely is it adherent to the umbilicus as a solid strand or pervious canal. It is evident that free mobility of the diverticulum is the most frequent cause of knotting and volvulus, but it sometimes forms a bridge under which intestines become incarcerated. This type of strangulation is generally the result of fixation of the diverticulum by peritonitis. The diverticulum is usually adherent to the mesentery, but it may also adhere to other portions of the intestines, the omentum, cæcum, small intestines, or to the pelvic organs, thereby possibly causing intestinal incarceration.

When the diverticulum is abnormally long and has a kind of club-shaped swelling at its free end, a not infrequent type of obstruction may occur. The diverticulum may form a sort of slipknot in which the ampulla, being the thicker portion, tends to tighten the knot (hence called by the French "*clef de l'étranglement*") (see Fig. 36). We pass over several other forms of obstruction which are described by Treves, as they are more infrequent.

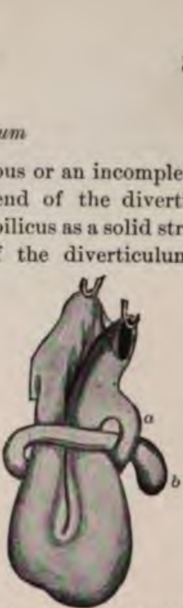

FIG. 36.—INTERNAL STRANGULATION OF A LOOP OF SMALL INTESTINE BY A MECKEL'S DIVERTICULUM COILED ABOUT IT.
a, attachment of the diverticulum to the intestine. *b*, its club-shaped end (Regnault-Béclard).

5. *Strangulation from Internal Herniæ*

According to Leichtenstern, internal herniæ are those which lie either entirely within the abdominal or thoracic cavities, or which are situated retro- or subperitoneally, parallel to the abdominal wall and protrude into the abdominal cavity, but never, even by continued growth, appear externally.

By external herniæ we mean those which push the peritoneum in an external direction, and which, by continued growth, become externally visible tumours. Leichtenstern, who has given us the most thorough description of this subject, distinguishes three forms :

1. Very small, external herniæ, which, particularly in fat individuals, may remain latent throughout.

2. Interstitial herniæ which run their course under the clinical picture of an internal hernia. They originate from the different hernial canals, most frequently from the inguinal. They occur either alone or in connection with external inguinal or femoral herniæ, whose subsidiary swelling they represent. Interstitial herniæ are generally the result of taxis and efforts at reduction. Interstitial incarceration has often been observed in the presence of mobility of the main hernia.

3. Finally, the following very rare herniæ—important only surgically and anatomically—run the course of the internal herniæ, viz., obturator, sciatic, perineal, rectal, vaginal, and lumbar herniæ.

With the exception of diaphragmatic hernia, true "internal herniæ" can hardly ever be diagnosticated. We therefore limit ourselves to the mention of their names, and again refer to the exemplary description of Leichtenstern. This author gives the following varieties of true internal hernia: hernia retro-peritonealis anterior (hernia intra-iliaca, ante-vesicalis, retro-pubica, hernia interna vaginalis testiculi, hernia iliaco sub-fascialis), hernia duodeno-jejunalis (Treitz's hernia, of which about 50 cases have been described), hernia pericæcalis, hernia intersigmoidea, hernia intra-epiploica, hernia ligam. uteri lati, hernia foramen Winslowii, and, finally, hernia diaphragmatica.

There are two forms of diaphragmatic hernia, the true and the false. The former name is applied to those cases in which an abdominal organ or organs have entered the pleural cavity through an opening in the diaphragm, and which have as hernial covering either pleura or peritoneum, or both. The latter name (false) is applied to those cases in which the abdominal viscera pass through an opening both of the diaphragm and of the contiguous serous membranes (pleura and peritoneum), the abdominal organs coming into direct contact with the thoracic. Such visceral displacements must be looked upon rather as a prolapse or ectopia than as a true hernia. Most diaphragmatic herniæ are "false." Of 254 cases, only 28 were true.

Entrance of the abdominal viscera into the chest cavity is caused either by congenital defects of the diaphragm, by a physiologically preformed fenestrum, or by an absence of continuity in the diaphragm from inflammation or (more often) traumatism.

Congenital defects occur most frequently in the muscular portion of the diaphragm, usually to the left side (in the proportion of 98 to 19), and occasionally extend over an entire half of the diaphragm. Acquired diaphragmatic herniæ are also generally leftsided; the place of entrance is more frequently in the posterior than in the anterior portion of the midriff.

The preformed openings in the diaphragm which may give rise to herniæ are (1) the esophageal opening; (2) the foramen of Morgagni (in that portion of the midriff corresponding to the sternum and the seventh costal cartilage of either side); (3) the foramen of Bochdalek (posteriorly between lumbar and costal division of the diaphragm); (4) the point of entrance for the sympathetic nerve (between the external and median crura).

The abdominal viscus most frequently displaced is the stomach; then follow the transverse colon, omentum, small intestines, spleen, liver, pancreas, and kidney. The greater curvature of the stomach is regularly found uppermost, and the lesser curvature below. If the fundus alone be involved, and not the pylorus, volvulus with incarceration of the stomach may follow. Two or more organs are generally displaced. When only one viscus is displaced it is usually the stomach, less frequently the colon, small intestine, or omentum. Most cases of diaphragmatic hernia are accidentally discovered at the autopsy. They sometimes produce significant symptoms, which under favourable circumstances may lead to a diagnosis during life. Leichtenstern[44], who first successfully recognised a case of diaphragmatic hernia, considers that the diagnosis may be made if pneumothorax can be excluded, and if it can be proved that there are in the thoracic cavity other air-containing organs, which upon auscultation and percussion present differences depending upon their varying conditions of fulness.

Filling up the stomach or colon with air or water may aid the diagnosis. Displacement of the heart to the right is also of diagnostic significance (P. Guttmann[45] and Abel[46]). Finally, trans-illumination of the stomach, particularly, its radiograph (by the introduction of a soft metallic sound), ought to make diagnosis possible.

SYMPTOMATOLOGY

Before discussing the individual symptoms of these forms of obstruction we must mention several facts of great practical importance.

First of all as to the seat of incarceration. In the overwhelming number of cases the ileum is the segment affected; the other intestinal segments, both above and below the ileum, are so rarely affected that they hardly come up for consideration. The cause of this frequency of ileal incarceration lies in its anatomical position, and in the possibility of its coming into contact with all those other organs which are most apt to be incarcerated (mesentery, great omentum, Meckel's diverticulum, vermiform appendix, pelvic organs, hernial canals, etc.).

The age requires consideration. Though incarceration may occur at all ages, yet statistics show that it is essentially a disease of early life, that it is most frequent in the second decade, and that it occurs only exceptionally in the later periods, or in the first decade.

Sex is also a factor, though the difference in this particular is not sufficiently marked to allow of positive data to be based thereupon. In women the puerperium and the diseases incidental thereto are considered as predisposing factors. We find that appendix affections, herniæ, traumatism, and Meckel's diverticulum act more frequently as predisposing factors in the male than in the female, so that the proportion of incarcerations is, according to Leichtenstern, 180 males to 118 females.

In its important details the clinical picture of incarceration coincides with that already given under general symptomatology. While referring to the latter we again point out certain important symptoms which are particularly prominent in the clinical *ensemble*. Thus we would mention the acute onset in the midst of normal health or after a slight illness, or occasionally after a traumatism, the violent development of symptoms finally leading to death, the severe pain, the vomitus rapidly becoming feculent, the complete retention of stool and gases and contrasting strangely with the absence or only slight presence of meteorism, the retention of urine, rapidly increasing debility, collapse amid complete consciousness, and the absence of fever when the case is not complicated by peritonitis. The few exceptions to this type are characterized by a less violent development of symptoms. The course of the disease may be less sudden and show deceptive remissions. These differences mainly depend upon the form and degree of the obstruction.

DIAGNOSIS

Not only the presence of an incarceration or strangulation, but also its location and nature must be determined. Occasionally, favourable circumstances—a history which points directly to the source of obstruction—allow of a correct diagnosis being made, but in the large majority of cases the diagnosis is impossible. If we have diagnosticated internal strangulation, we may assume the site of the obstruction to be in the lower ileum, since that portion is more frequently involved. Regarding the differentiation of this (internal) from other forms of obstruction, we shall only mention the most important points which enable us to recognise the condition in favourable cases: early period of life, a history of inflammatory processes in the abdominal cavity or of the pelvic viscera, previous traumatism, previous laparotomy, very acute onset of severe symptoms, absence of tumour, *absence of marked meteorism, the presence of a fixed, inflated coil of intestine and perhaps visible peristalsis*, hemorrhagic exudate, and the absence of bloody stools. (Compare in this connection the differential diagnosis, page 407.) When the case is complicated by peritonitis the diagnosis, as a rule, cannot be made.

II. Volvulus

Preliminary Observations.—By volvulus we understand a torsion of the intestine about its mesenteric axis, or the knotting together of two coils of gut. We must consider volvulus of the sigmoid flexure (the most frequent and practically the most important form), volvulus of the ascending colon, and volvulus of the small intestine.

Volvulus of the sigmoid flexure is found in two thirds of all the cases, and occurs when the sigmoid is very long, and its mesocolon long and narrow, so that the ends of the arch are brought nearer to each other (see Fig. 37). It is quite apparent that under such conditions volvulus, with the mesentery as the axis, may easily occur.

The causes of volvulus are direct and indirect (predisposing). Of the latter the following may be mentioned: congenital predisposition (an abnormally long sigmoid with a long, narrow mesentery), habitual constipation, a purely vegetable diet (which, according to Lingen and Küttner, explains the frequency of volvulus in the peasants of Russia), and peritoneal inflammation in the neighbourhood of the sigmoid flexure, with consequent cicatrization of its mesentery (mesenteritis, see page 394). Trauma, marked natural or artificial intestinal peristalsis or its reverse, acute intestinal paresis, errors of diet, foreign bodies, gallstones, dysentery with consequent cicatrices, tumours, laparotomies, etc., are direct causes of volvulus. Undoubtedly fæcal impaction is the most frequent cause of volvulus. It occasions a sinking of the upper limb of the sigmoid flexure upon the lower, or the lower limb gradually approaches the upper till finally the volvulus is completed by severe peristalsis, induced perhaps by a strong drastic purge. In most cases there is a torsion of 180 degrees, more rarely of 360 degrees. The torsion takes place about the mesenteric axis, and also, to some extent, about its [intestine] own axis. In these cases either the descending colon lies in front of the rectum (type *rectum en arrière*, Potain), or the rectum lies in front of the colon (type *rectum en avant*) (see Fig. 38, A, B). The former type is by

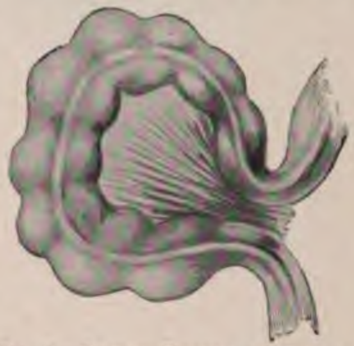

Fig. 37.—Sigmoid Flexure showing a Tendency to Volvulus Formation. [Treves.]

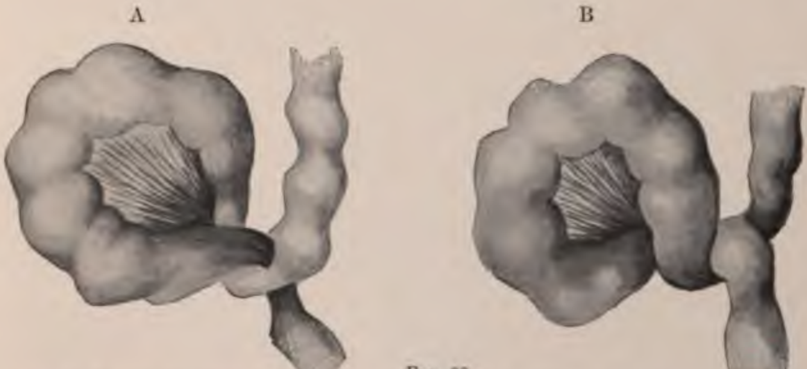

Fig. 38.

Type Rectum en Arrière. (Potain.)

Type Rectum en Avant. (Potain.)

far the more frequent. If the volvulus be complete (i. e., torsion of 270 to 360 degrees), spontaneous untwisting is impossible, because of the changes which soon occur in the affected limb of the sigmoid flexure (accumulation of blood, exudation, formation of gas), and in that lying above it (intense meteorism, intestinal paresis). According to experiments of Melchioris[47], the resistance of the abdominal wall also prevents untwisting. Finally, peritonitis will contribute to the fixation of the volvulus. Spontaneous untwisting may follow incomplete volvulus.

As already stated, volvulus of the other segments of the large intestine (cæcum, transverse colon) is extremely rare. The conditions under which it occurs are similar to those of volvulus of the sigmoid. Somewhat more frequent, though still quite rare, is volvulus of single or several coils of small intestine. The amount of torsion is generally about 180 degrees. Usually the upper end of the small intestine lies below and to the left, and the lower end above and to the right. The intestines and mesentery lying to the right are transposed to the left, and *vice versa*. The intestinal obstruction need not be complete. Volvulus of this kind occurs even in earliest childhood. According to Leichtenstern[1], congenital malformation of the mesentery, in which ileum, cæcum, and ascending colon have a common mesentery, seems particularly to predispose to volvulus. Volvulus of the small intestine occurs most frequently in coils which have for a long time been either in a large hernial sac or in its vicinity, or which have become adherent to pelvic viscera (Leichtenstern).

We have mentioned that the volvulus usually occurs about the mesenteric axis of the bowel. Besides this there is a true torsion about the intestinal axis; such cases generally affect the large intestines, particularly the cæcum and ascending colon. Leichtenstern considers most of them as kinking following displacements. These volvuli need not produce complete obstruction; this may only occur when other changes are superadded—for example, when the mesentery of a coil of small gut is thrown across the place of kinking.

Finally, we must briefly describe the knotting together of two intestinal coils. The sigmoid flexure and the ileum are most often twisted about each other (see Fig. 39). Much more rarely there is knotting or coiling together of small intestine, or a twist of the jejuno-ileum, cæcum, and ascending colon. In the first-mentioned instance, according to Leichtenstern, the most frequent occurrence is the displacement of small intestine across the narrow mesentery of the sigmoid. Through the space which is thus formed by the posterior abdominal wall and the roots of the crossed coil of small intestine and sigmoid flexure, the superior portion of the sigmoid enters, after passing from below upward in front of the small intestine. The clinical course of this knot formation is exceedingly acute; it is fatal in one or two days. Such formations are frequently ushered in by violent diarrhœas, which may remain pronounced throughout the course of the disease, to which they lend a certain similarity to cholera.

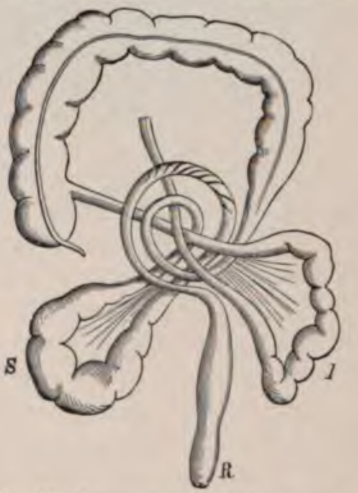

FIG. 39.—SCHEMATIC DRAWING TO ILLUSTRATE A KNOTTING TOGETHER OF ILEUM (*I*) AND SIGMOID FLEXURE (*S*). *R*, rectum. (Leichtenstern.)

SYMPTOMATOLOGY

In what follows we shall describe more especially the clinical symptoms of sigmoid volvulus. Because of their rarity and the obscurity of their symptoms the other forms have but little clinical interest, and will be very briefly described at the end of this section. According to most clinicians, volvulus is a disease of late life (forty to sixty years). In 20 cases Treves[22] found the average age forty-nine years. In his statistics Leichtenstern[1] found only one case in the first decade. Men seem to be more predisposed to this affection than women; Treves gives the proportion as 4 to 1. The occurrence of volvulus late in life is explained by the fact already mentioned (page 377), that it develops most frequently in connection with habitual constipation, which is most severe in the fourth to the sixth decades of life. In view of the fact that obstinate constipation is so frequent in women, it is rather striking that the majority of cases of volvulus should occur in men. Perhaps more extensive statistics will show that the female sex is after all more prone to this affection.

Except for occasional acute attacks, the varieties of intestinal obstruction already described generally present no evidences of previous intestinal disease; chronic constipation really forms the initial stage of an affection which finally ends with an attack of volvulus.

The pernicious character of the constipation is often apparent from the history. The constipation increases steadily, and increasing doses of drastic purges produce only incomplete evacuations of a spastic or diarrhœal character, accompanied by severe pain and perhaps by nausea and vomiting. After a longer or shorter period volvulus suddenly occurs.

As in other varieties of intestinal obstruction, the main symptoms are pain, vomiting, complete intestinal occlusion, disturbance of general health. Excepting that they are less violent, all these symptoms differ but little from those of obstruction of the small bowel.

Pain, the first and most striking symptom, is quite marked, but is not so severe or continuous as in incarceration or strangulation. It is intermittent, and is relieved by small doses of opium. The patient may be able to obtain sleep without narcotics, and otherwise present a certain euphoria. The pain is usually most pronounced about the umbilicus, more rarely it is found in the vicinity of the sigmoid itself. Tenesmus may accompany the pain, but even in well-defined cases of volvulus it may be absent. If peritonitis sets in, the pain is increased and becomes more diffuse.

Sensitiveness to pressure may be absent in the beginning of the affection; later, the tenderness becomes easily demonstrable, and is generally localized in the umbilical region. Marked increase in the degree and area of this sensitiveness to pressure is always a suspicious sign of beginning peritonitis.

In the beginning, the vomiting is reflex and accompanies the paroxysms of pain. Occasionally vomiting is entirely absent, and then nausea or severe explosive eructations occur, followed by momentary relief. The vomitus is by no means very copious; at first it consists of the stomach contents mixed with mucus, later, of grayish green or brownish masses with a slight "intestinal odour." As stated by Treves,* the vomiting is very rarely stercoraceous. If in the presence of well-defined symptoms stercoraceous vomiting occurs quite early, it always indicates an unfavourable termination of the disease.

As in the other forms of intestinal obstruction, anorexia and very severe thirst occur also in volvulus.

Usually, from the very beginning of the disease neither fæces nor flatus are passed. Small quantities of fæcal masses come away with the enemata or rectal irrigations, but, as already mentioned, no flatus accompanies such evacuations. If purges or enemata do

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[* Loc. cit., p. 299.]

produce stools accompanied by flatus, incomplete volvulus should be suspected. A single evacuation of this kind should not make the physician too sanguine, for experience has shown that an incomplete volvulus may readily become complete through peristalsis. Therefore the general condition of the patient is more significant than the condition of the stool. Occasionally blood has been found in the evacuations.

Local tympanites is a very important and characteristic symptom of volvulus of the sigmoid; in no other form of intestinal obstruction is this symptom so prominent from the very beginning of the disease. The meteorism is not limited to the neighbourhood of the sigmoid flexure; on the contrary, this latter region is generally occupied by coils of small intestine. As previously mentioned (page 23), the tympanites extend toward the right in front and upward. Curschmann[48] has made the important observation that in this disease one may find tympanitic intestinal coils over the entire abdomen, but not at the site of the volvulus—i. e., in the vicinity of the sigmoid. Here von Wahl's symptom of distended intestinal coils may be very well demonstrated (page 363). If peritonitis develops, the meteorism gradually becomes general, and it is then impossible to determine the location of the volvulus. When the meteorism is moderate, we may, in the lower abdomen, at or to the right of the median line, occasionally palpate a resilient tumour having the resistance of a tightly distended air cushion. The tumour usually extends from the lower left side upward toward the right hypochondrium; there may be tympanitic or metallic tinkling on percussion, but there may also be dulness if the sigmoid flexure is very edematous, or contains large quantities of fæces.

Visible peristalsis is rarely observed. Treves noticed this phenomenon twice in 20 cases. Incomplete occlusion is probably present in such cases.

In volvulus of the sigmoid flexure the general condition of the patient also suffers; the reaction, however, is not as severe as in obstruction of the small intestine. The pulse remains of good quality for a long time, and is not immoderately frequent, the face does not bear that collapsed expression (facies hippocratica) that is seen, for instance, in strangulation of the small intestine. In one of my cases, a woman of sixty-four years, three days after the onset of symptoms of obstruction, the patient was able to descend two flights of stairs without apparent effort. If diffuse peritonitis or perforation occurs, the symptoms very rapidly change.

The clinical picture of volvulus of other segments of the large intestine, and of the small intestine, is somewhat different from that of the sigmoid flexure. For example, in volvulus of the upper portion of the large bowel, both tenesmus and bloody evacuations are absent and the pain extends more toward the back. In volvulus of the small bowel, on the other hand, the segment below the point of constriction may contain intestinal contents, and these may be evacuated. A case described by Naunyn* was characterized by the passage of large quantities of unaltered blood. The meteorism naturally is variously localized. A case of Nothnagel's † demonstrates that the course of the disease is not necessarily violent, and may for a time appear favourable.

DIAGNOSIS

In diagnosticating volvulus of the sigmoid flexure, the history and the subjective and objective symptoms require consideration.

The *history* will give valuable data. It informs us regarding the existence of long-standing chronic constipation, and perhaps also of a former incomplete attack of volvulus of the sigmoid flexure.

The *subjective symptoms* to be considered are the pain, the vomiting, the complete intestinal obstruction, and the general condition of the patient. These have all been already described. Collectively they indicate an intestinal obstruction. The manner of their occurrence, which, as already mentioned, is characterized by a certain benignancy and slow development, offers serviceable hints regarding the nature and the seat of the obstruction.

The most important *objective* sign is local tympanites. If well developed, if the sigmoid flexure can be differentiated from other intestinal coils by auscultatory percussion, if the distended sigmoid flexure can be grasped and distinctly palpated, and if the other symptoms above mentioned are present, the diagnosis is generally assured. If only small quantities of air or water can be injected into the rectum, and these are immediately returned, the diagnosis is made still more certain. On the other hand, as Treves correctly observes, the possibility of large quantities of injected water being retained by the lowest bowel segment by no means militates against occlusion of the sigmoid flexure. It seems to me important also that injections of large quantities of water cannot produce splashing sounds in the cæcum or transverse colon. In the presence of severe general tympanites the demonstration of splashing sounds in the cæcum may not be possible, even though the sigmoid flexure be pervious.

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* Loc. cit., p. 110.

† Loc. cit., p. 351.

The indican test may be used as a diagnostic aid. Indican, if absent, or present only in very small quantities, speaks for obstruction of the large bowel. Free hemorrhagic fluid has sometimes been found in operations for volvulus, but with marked tympanites its recognition is extremely difficult.

It is seldom possible to diagnosticate volvulus of the upper segments of the large or of the small intestine.

III. Invagination, Intussusception

Preliminary Remarks.—Invagination is a condition in which a portion of the intestine is pushed or inverted into the lumen of that adjoining. Thus, three tubes are telescoped into one another (see Fig. 40). The outer tube is termed the intussuscipiens or sheath, and the two inner, which are generally full of folds, the intussusceptum or invaginatum. The latter is divided into the returning (external) and the entering (internal) tubes. The entering tube lies against the sheath at the "neck" of the invagination and is continuous with the returning tube at the lower (free) end of the intussusceptum. The point of junction of the two internal layers is known as the apex. Its relation to these layers is fixed; with them it always advances farther into the intussuscipiens. The mucous surfaces of the outer and middle tubes and the serous surfaces of the inner and middle tubes are opposed to each other. The mesentery of the gut is invaginated with it, and since the mesentery is compressed and dragged upon by the outer layer, the intussusceptum becomes concave at its mesenteric border, and hence is pulled eccentrically, and not axially, toward the intussuscipiens.

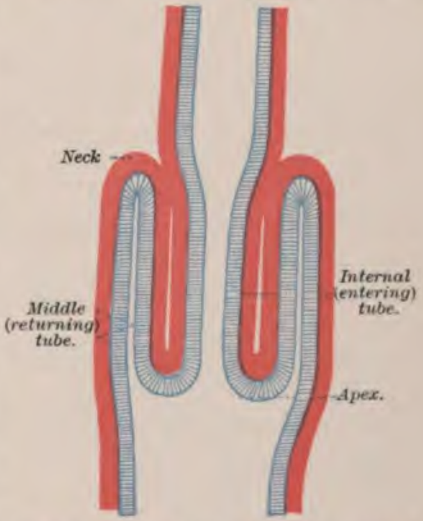

FIG. 40.—SCHEMATIC DRAWING TO ILLUSTRATE A SIMPLE INTESTINAL INVAGINATION.

Besides the ordinary forms, double, or more rarely triple, invagination, occurs. In the former there are five, in the latter seven, intestinal tubes. It is necessary also to make a distinction between complete and incomplete invagination. In the incomplete form only a single portion of the intestinal wall projects into the lumen. Partial invaginations are sometimes found when tumours (generally benign in character) drag one or more coats of the intestines after them. As observations of Böttcher[9] and Fleiner[50] have shown, invaginations incomplete at the outset, may finally become complete. Several years ago I observed a partial intussusception in a successfully operated case of cancer of the cæcum. Besides simple invagination, D'Arcy Power, Birch-Hirschfeld, and Thomas have described cases in which two intussusceptions were present in different portions of the bowel. Such a condition is, however, extremely rare.

From an etiological standpoint we must distinguish two different types of invagination: the physiological and the pathological (Nothnagel), or the agonal and the vital (inflammatory) forms (Leichtenstern).

The first variety very probably occurs immediately preceding death. At that time one intestinal segment may lose its power of contracting before another; when an adjoining portion of the bowel then contracts an invagination of the second part into the lumen of that already paralyzed may take place. This form of invagination occurs almost entirely in the small intestine. There may be more than one such invagination. They occur more frequently in children than in adults, and are found both in an ascending and a descending direction. They are further distinguished from the pathological variety by the fact that the mesentery is never drawn into the invagination. This form cannot be diagnosticated before death.

In contradistinction to the above variety, vital or pathological invagination—the only form which is of practical importance—is generally single and often of considerable size. The invagination is almost always in a descending direction (Leichtenstern found only 8 ascending invaginations in 593 cases), and is constantly accompanied by invagination of the mesentery.

Intussusception may occur in all segments of the large and small intestines, but with very varying frequency. In general, we distinguish *invaginatio enterica* (small bowel into small bowel), *invaginatio ileo-cæcalis* (small bowel into large bowel), and *invaginatio colica* (large bowel into large bowel). Special subdivisions are: *invaginatio ileo-duodenalis*, *duodeno-jejunalis*, *jejunalis*, *jejuno-iliaca*, *ileo-colica*, *iliaca-ileo-colica*, *colica*, *colica-rectalis*, and *rec-*

talis. The most important variety is invagination of the small into the large intestine (see Fig. 41), which, according to Leichtenstern, from an analysis of 479 cases, occurs in 52 per cent of cases at all ages. During the first year of life the percentage is as high as 70 per cent. Next in frequency are iliac (30 per cent) and colic invaginations (18 per cent). The proportion is different in adults, for the iliac and ileo-cæcal varieties are of about equal frequency. In invagination of the ileum the lower portion of this part of the bowel is usually affected. Colic invaginations are more frequent in the descending colon and in the sigmoid flexure than in the other portions of the large bowel.

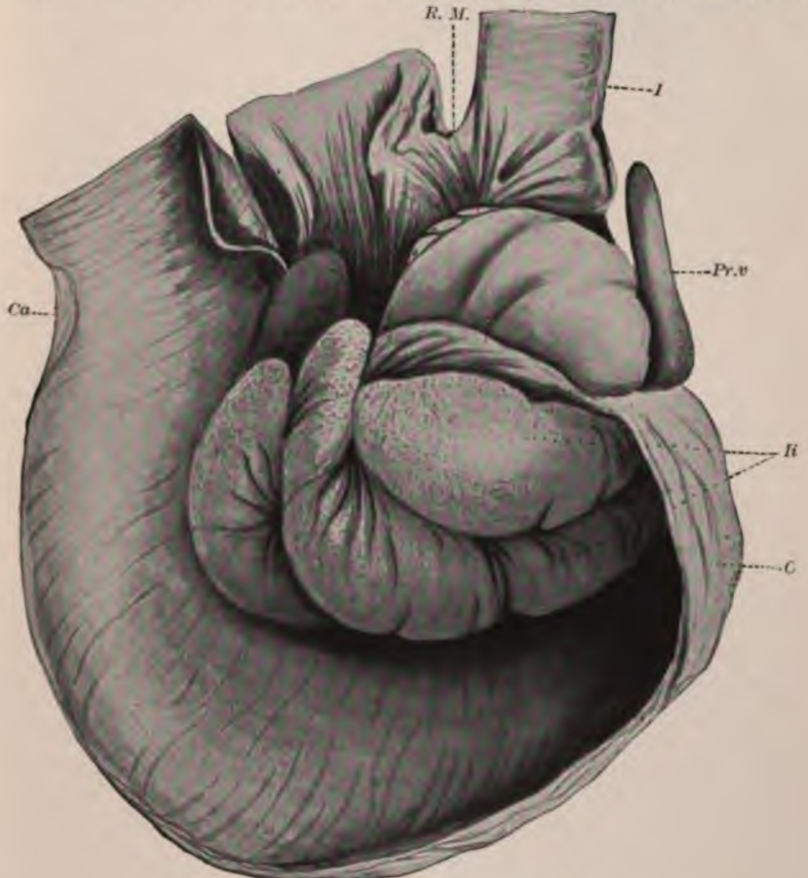

FIG. 41.—ILEO-CÆCAL INTUSSUSCEPTION.

I, ileum; *Ii*, invaginated ileum; *C*, cæcum; *R. M.*, mesenteric base; *Pr.v*, vermiform appendix; *Ca*, ascending colon. (Taken from the collection of Prof. Langerhans, of Berlin.)

Invagination occurs most frequently in childhood. According to Leichtenstern, one half of all intussusceptions occur during the first decade. In the first year of life, and particularly between the fourth month and the end of the first year, invaginations are very frequent.

Regarding sex, the majority of cases occur in males. The *chronic* forms are found most often between the twentieth and fortieth years of life (50 per cent); then follows the first decade with a frequency of 25 per cent. According to Raffinesque[51], in 51 cases of *chronic* invagination, 38 occurred in men and 33 in women.

At the present day opinions still differ regarding the etiology of intussusception. Contrasted with each other are the spasmodic (Dance, Cruveilhier, Beriton, Bristowe, Raffinesque, Nothnagel) and paralytic theories, the latter upheld mainly by Leichtenstern. According to the former theory an energetic, circular tetanic contraction of an isolated portion of the intestine constitutes the starting point of the inversion. This occurs in such a manner "that the intestinal segment below and immediately adjoining the spastically contracted portion is drawn up over the latter" (Nothnagel). Leichtenstern, on the other hand, claims that the bowel segment in question becomes paralyzed through certain intercurrent circumstances (diarrhœa, ingesta, traumatism, partial peritonitis); this segment is then everted and becomes invaginated with the entering internal contractile bowel lying below. The latter forms the vaginal portion of the intussusception. Again, D'Arcy Power[52], who has won renown because of his work in the pathology and operative treatment of intussusception, believes that a disproportion between the width of the ileum and cæcum is the true cause. If either congenitally or otherwise the circumference of the cæcum is considerably increased, a predisposition to invagination occurs. The discussion of these hypotheses, which are thoroughly described by Nothnagel, Leichtenstern, and Treves, would lead us too far. Animal experiments and theoretical considerations incline me toward the spastic theory.

The direct causes of invagination, according to the statistics of 593 cases gathered by Leichtenstern[53], do not appear to be uniform. In 111 apparently healthy individuals the disease began suddenly; in the remaining number of cases the following etiological factors were found: intestinal polypi (30 cases), intestinal cancer and stricture (6 cases), diarrhœa (21 cases), other abnormal intestinal functions (25 cases), ingesta (28 cases), abdominal contusion (14 cases), concussion of the body (12 cases), invagination during pregnancy or puerperium (7 cases), "catching cold" (6 cases), various acute and chronic diseases as well as indifferent and doubtful factors (66 cases). From these statistics we can only conclude that we are absolutely in the dark regarding the real causes of intussusception.

Symptomatology

The symptoms of intussusception are those of a severe intestinal obstruction with all its characteristics. Because of their peculiarity, the pain, vomiting, character of evacuations, the condition of the abdomen, and the tumour formed by the invaginated bowel, must be described in detail.

Spasmodic pain, the first and most prominent symptom, generally appears quite suddenly—in nurslings while at the breast, in older children during play, in adults in the midst of work or perhaps at night. From the beginning the pain is usually of an extremely threatening character, so that in children collapse or convulsions may usher in the disease, while in adults the severity of the pain causes the patient to writhe in agony. After the initial paroxysm—which is probably caused by incarceration of the mesentery—the pain may become continuous, or, as is often the case, may cease for one or several hours. During the intermissions the patient may take some nourishment and for a very short time feel comparatively well. The pain may cease before death in consequence of paralysis of the pain centres, but there are many cases in which the pain continues till death. If the acute invagination becomes chronic, the pain may take on a marked paroxysmal character, just as has been described in intestinal stenosis. The site of the pain depends upon the part of the bowel affected, and varies considerably. In children it is generally limited to the region of the umbilicus. In adults the pain may be localized in a portion of the intestine which corresponds fairly well with the seat of invagination; this fact can be of diagnostic importance. The pain is generally accompanied by distressing *tenesmus*. The character of the evacuations (to be described later) and the tenesmus may at first view present a striking similarity to that of acute dysentery. The tenesmus is much more severe in children than in adults, so that paresis of the sphincters very soon results. The higher up in the intestine the invagination the less marked is the tenesmus, and *vice versa*.

In childhood, *vomiting* accompanies the pain from the onset, and may very rapidly run through all the various stages (already described) up to feculent vomiting. In adults, vomiting is by no means as constant as in the other forms of intestinal obstruction. It may be absent throughout or may occur at certain intervals, or, as in children, it may be very violent and continuous. These various characteristics depend upon the greater or lesser completeness of the obstruction, upon the amount of the mesentery invaginated and the degree of its compression, and upon the site of the invagination. Unfavourable conditions are found in the iliac, ilio-cæcal, and ileo-colic forms, while invaginations of the large bowel generally run a comparatively mild course. As in intestinal stenosis, vomiting is least prominent in the chronic forms.

The *character of the evacuations* is one of the most important objective symptoms. Invaginations are distinguished from most other forms of intestinal obstruction in that evacuations do not immediately cease, but one or more stools, evidently derived from the distal intestinal segment, may be passed after the onset of the intussusception. If real stools no longer occur, there may be repeated evacuations of blood, blood and mucus, blood and pus, or of gangrenous masses. In these cases the tenesmus is apt to be very severe. The *hemorrhages constitute one of the most constant symptoms; they are absent in only 20 per cent of acute cases;* they vary in amount according to the site and extent of the invagination. As soon as the process becomes subacute the hemorrhages may cease or temporarily disappear. On the other hand, large, gangrenous, putrid pieces of intestine may be passed per rectum. In chronic invagination, hemorrhage, although much more frequent than in other forms of chronic intestinal stenosis, may be entirely, or almost entirely, absent. Rarely is the hemorrhage large in amount. The character of the evacuations varies very much in chronic invaginations, and scarcely two cases are alike.

Aside from the tumour the abdomen presents no noteworthy changes. *Tympanites* is rare; when present, it is not well marked. It is least when diarrhœa is present, somewhat more marked with absolute constipation, and extensive only when peritonitis supervenes.

Formerly the presence of a symptom known as the "signe de dance" was considered important. It was said to consist in a depression in the right inguinal region or in the right iliac fossa, presumably caused by displacement of the cæcum. This symptom has lost all value after Raffinesque[51] showed that even in chronic invaginations—those most favourable for this sign—it is present in only about 4 per cent of the cases.

The *invagination* tumour is of much greater importance, and may even be pathognomonic. It may be palpated through the abdomen, or through the rectum or vagina. It is present in about half of all acute cases, but it can be more easily found and palpated in chronic cases. The tumour is, however, not palpable with uniform frequency in all varieties of invagination. According to Treves, it is most often felt in intussusception of the ileo-cæcal region and of the cæcum, least so in that of the small intestines and in the ileo-colic form. The tumour is more clearly demonstrable in children than in adults. This is due to the fact that in the former, because of the softer abdominal walls, tumours can be more easily felt. As Henoch[54] states, even in such favourable cases the tumour may be obscured by distended coils of intestine. The tumour may vary in size from a hen's egg to that of the adult forearm. It is smooth, moderately hard, of varying consistency, sausage-shaped, and somewhat curved. The smaller tumours are the more frequent. The size of the tumour that can be mapped out by palpation does not always correspond to its real extent, for portions may be obscured at the flexures of the colon. The tumour is found most frequently over the ascending, next over the transverse colon. When lying in the cæcal region it indicates an ileal invagination. The invagination tumour, like all other intestinal tumours, is characterized by relatively great mobility. It is therefore very difficult, from the position of the invagination tumour, to correctly diagnosticate the original seat and kind of invagination. Provided it is not fixed by adhesions, the tumour can be moved from without, sometimes even to a very marked degree. It may temporarily disappear, and therefore Treves's warning, never to diagnosticate the absence of a tumour unless the abdomen is examined during a paroxysm of pain, is timely. Examination at the height of an attack of colic offers the best opportunity for distinctly palpating the tumour, and, at the same time, determining the existence of tetanic intestinal rigidity. The latter is rarely found in acute invagination; in chronic forms it is quite readily demonstrable.

In acute as well as in chronic cases there may be prolapse of the invagination tumour through the rectum. It generally occurs in acute invagination, and is most frequent in the ileo-cæcal and colic forms. When prolapsed, it may be directly palpated, and is seen as a hyperæmic or partly gangrenous tumour. Its origin is evident by the appearance at one point of the ileo-cæcal opening, and next to it a second opening, that of the appendix. Simple as is the demonstration of a prolapsed invagination tumour numerous errors have been made. For instance, invagination tumours have been mistaken for prolapse of the rectum, for polypi, or for hemorrhoids, and have been excised. Treves reported several remarkable instances in which, despite these operative errors, cures resulted. The reverse has also occurred, viz., that other tumours (a false diverticulum, at another time a blood coagulum) have been mistaken for an invagination tumour. Such confusion can generally be avoided by careful and repeated examination, particularly under narcosis.

DIAGNOSIS

Of all forms of intestinal obstruction, *acute* intussusception offers the most favourable opportunities for early diagnosis.

Among the *subjective* symptoms we must consider the sudden onset in the midst of good health, the immediate occurrence of intense pain of a convulsive or intermittent character, and the vomiting (by no means so violent as in other forms of obstruction, and feculent in only about 25 per cent of all cases). Tenesmus, present in about 50 per cent of all cases, is of special diagnostic significance. As, of all varieties of obstruction, it is found in volvulus of the sigmoid flexure alone, and here relatively seldom, I believe that well-defined, tenesmus is one of the most positive subjective symptoms of invagination. Disturbances of general health vary so much according to the age of the patient, the site, the special type, and the condition of the invagination, that they need scarcely to be considered in a diagnostic connection.

The most important of the *objective* signs is the presence of a tumour. As already mentioned, a tumour is demonstrable only in about one half of the cases. In obscure forms we would strongly recommend repeated examinations, particularly during a paroxysm of pain. At this time the invagination tumour approaches the anterior abdominal wall. By careful consideration of the remaining symptoms the character of the tumour will scarcely ever remain unrecognised.

If the tumour projects from the rectum, its peculiarities will immediately point to the diagnosis. The character of the evacuations is also a significant and possibly diagnostic phenomenon. Absolute constipation is generally absent. In addition, there is the frequency of bloody evacuations, or evacuations consisting of blood and mucus, or of pus mixed with gangrenous shreds. As already mentioned, bloody evacuations also occur in incarceration and strangulation, but the passing of purulent, gangrenous masses is typical of acute intussusception. When we suspect intussusception, early examinations of the stools and search for small microscopical amounts of pus should be made; such examinations may clear up an otherwise obscure clinical picture. For further diagnostic data see chapter on Differential Diagnosis.

The diagnosis of *chronic* intussusception is often quite difficult. Raffinesque[51] mentions that in 55 cases collected by him the diagnosis was incorrect in no less than 27. Here, again, pain is the most important and occasionally the most valuable subjective symptom. It is so often markedly intermittent (coliclike) in character that it may indeed be taken as the type of colic pain. Accompanying the pain, intestinal rigidity with all the characteristics of the tetanic contractions of intestinal stenosis (see page 356) generally occurs. The patients themselves are conscious of this rigidity.

Vomiting is not a reliable diagnostic symptom of chronic invagination. Its frequency and degree vary considerably. The stools present nothing characteristic. As Treves * remarks, "the only certain feature in the state of the bowels in chronic invagination is the feature of uncertainty." Of importance in this connection is the experience of Raffinesque[51], that diarhœa is present in about one half of all chronic cases. I have found no reports in medical literature concerning the presence of pus in the evacuations; such a condition is without doubt more rare than it is in the acute form.

Of the *objective* symptoms the most important is the invagination tumour. When well marked it is of greater diagnostic value than any other symptom. Regarding the nature of the tumour, we refer the reader to the section on symptomatology. In chronic intussusception the invaginated bowel not infrequently extends to the rectum (according to Raffinesque in about one third of the cases) and may be felt there. This is rare in the beginning, but more frequent in the later stages of the disease. Just as in acute cases, meteorism is not well marked, and the course of the affection and general condition of the patient present no special diagnostic characteristics.

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* Loc. cit., p. 100 [English edition, p. 420].

IV. Bending, Kinking, Adhesions, Mesenteric Contractions, Compression

In previous sections we have described those forms of obstruction caused by clefts, fenestra, false bands, etc., which may produce severe incarceration or strangulation. There is, however, another group, more infrequent and clinically less severe, in which intestinal obstruction may be produced by isolated peritoneal adhesions. They are distinguished from the above group in that both small and large intestine are affected with equal frequency. The same is true of compression of the bowel from without by other conditions (e. g., new growths).

Treves,* who has carefully described these forms of obstruction, distinguishes the following varieties:

1. *Obstruction over a Band*

"If several coils of a thin India-rubber pipe, through which water was flowing, were thrown over a tightly-drawn wire, the lumen of the tube would become more or less completely occluded at the spot where the wire was crossed" (Treves).

Very similarly we may imagine that if one or more coils of intestine are drawn across a taut tissue strand, the intestinal lumen will be narrowed. Through irregular peristalsis and partial adhesion of the coil to the band more favourable conditions for such an occurrence are created. Treves discovered only 4 of these cases.

2. *Obstruction from Acute Kinking Due to Traction upon an Isolated Band or an Adherent Diverticulum*

In this instance a band attached to the bowel so drags upon its point of attachment that the intestine is acutely bent at the latter point, and is finally completely occluded. This condition is most frequently met with in Meckel's diverticulum or in isolated bands connected with the ileum. The ileum, because of its short mesentery, is particularly predisposed to kinking. Owing to its tendency to displacements, the large intestine may also become kinked or bent.

3. *Obstruction from Adhesions which retain the Bowel in a Bent Position*

The site of these abnormal bands is either the abdominal or the pelvic wall or the abdominal organs, as the liver, kidneys, and spleen. The etiological factors are traumatism, pelvic peritonitis, perityphlitis, incarcerated and reduced herniæ, etc. The kinking may be single or multiple—a fact to be remembered at laparotomies. The kinking may give no symptoms during life, may partially obstruct the passage of the fæces, or, in consequence of some external or internal influence, may suddenly cause all the symptoms of an acute intestinal obstruction. The forms of intestinal obstruction so frequently observed after reduction of femoral herniæ are striking examples of this variety.

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* Loc. cit., p. 100, etc. [English edition (1899), p. 75, etc.].

4. *Obstruction by Means of Adhesions of Intestinal Coils to Each Other*

This may occur both in the small and large intestines. In the former it is most frequent with herniæ. If, for example, a large coil is markedly compressed in the neck of the hernial canal, adhesions develop at this point; after reduction of the hernia these remain. Only the portions of the loop that were compressed become adherent (open loop). In small incarcerated herniæ, on the contrary, the entire loop of the bowel in question is bound together by adhesions (closed loop). Similar adhesive bands occur after intestinal ulceration with consecutive local peritonitis, or, as Treves specially points out, as the result of cheesy degeneration of mesenteric glands.

Adhesions between coils of the large bowel occur particularly after displacements or ulcers of this portion of the intestine. Displacements have been discussed in detail in a previous chapter (see page 255). From these changes in position accumulations of fæces occur and catarrhal changes easily develop; stercoral ulcers may result, and cause local peritonitis and adhesions to the adjoining part of the bowel or other abdominal organs.

In recent years Kelling[55] and Westphalen[56] have shown that adhesions between the transverse colon and the liver occur and cause a number of intestinal disturbances (pain accompanying peristalsis).

5. *Obstruction due to Traction upon the Intestinal Wall by a Diverticulum*

Treves has called attention to a stricture which is characterized by marked narrowing of the small intestine and by numerous ulcerations of the mucous membrane above the stricture. Complete intestinal obstruction may here result from distortion of the bowel wall by a diverticulum.

6. *Narrowing of the Bowel from shrinking of the Mesentery after Inflammation*

The affection first described by Virchow under the name of *peritonitis chronica mesenterialis* apparently plays a much greater part in the etiology of intestinal obstruction than was formerly supposed. In one year, for example, Riedel[57] observed no less than 8 cases resulting from such cicatrization. The usual site of mesenteric inflammation with subsequent cicatricial contraction is the sigmoid flexure, where, as already stated, it may lead to volvulus. This process also occurs at the cæcum, and, as Riedel has recently shown, in the mesentery of the small intestine as well as in the peritoneum of the posterior abdominal wall. Besides volvulus of the sigmoid, mesenteric contraction causes a displacement through traction of some part of the intestines and disturbances of intestinal mobility; such disturbances may present the picture of chronic bowel stenosis, or in extremely severe cases they may lead to acute intestinal obstruction.

7. *Compression of the Bowel from Without*

This term, in its narrowest sense, means the pressure produced in an intestinal segment by a body adjoining it; such pressure either narrows or completely obliterates the intestinal lumen. Compression is generally caused by malignant or benign neoplasms, which may belong to the most varied organs (stomach, intestines, liver, pancreas, spleen, kidney, lymph glands, mesentery, pelvic bones, uterus, ovaries, etc.). A tumour originating in the intestines may compress a neighbouring segment.

Besides neoplasms, other pathological conditions may compress the bowel from without; for example, a retroflexed uterus, large vesical calculus, peri- and paratyphlitic abscesses, floating spleen and floating kidney, tumours of the pancreas from hemorrhage or cysts, etc.

Owing to its situation, the rectum is most often pressed upon, usually by pelvic tumours. According to Leichtenstern, this occurs in 60 per cent of all cases. Then follow in order of frequency the sigmoid flexure, descending colon, the lower portion of the ileum, duodenum, and, finally, the ascending colon and hepatic flexure, middle portion of the ileum, and transverse colon.

In this connection it is important to remember the peculiar compression of the small intestines (duodenum or ileum) recently described by Schnitzler[58]. It is produced by the mesentery of coils of the small intestine which have descended into the pelvic cavity. In a similar manner, from traction of the pylorus in consequence of extreme gastrectasia and by compression of the duodenum and other portions of small intestine, the clinical picture of acute incarceration may be produced (L. Meyer[59]).

SYMPTOMATOLOGY AND DIAGNOSIS

The clinical symptoms of the above forms of intestinal occlusion possess, on the one hand, the character of stenosis, and, on the other, that of obstruction, and both conditions may suddenly or slowly interchange with the other. The few differential diagnostic signs described in the literature of the subject are not sufficient to distinguish these forms from other similar ones. We shall therefore not give any detailed account of them. Aside from several marked instances (obstruction after hernial reduction), one fact deserves mention—the course of the disease is generally milder and slower than the forms previously described.

Under certain conditions the *diagnosis* of these affections may be made, i. e., where the cause of the occlusion is visible or palpable (e. g., tumours, palpable adhesions), or where the history points directly to the nature of the disease (obstruction following hernial reduction, local peritonitis following traumatism, appendicitis with adhesions, previous operations, etc.). The fact that these forms of obstruction affect adults rather than children, and also that, as above stated, the symptoms are generally less severe than in strangulation, incarceration, or volvulus, may sometimes possess diagnostic importance. The exceptions are so numerous, however, that in a given case the last-mentioned data must be cautiously taken into account.

V. Internal Intestinal Stricture

Internal strictures are produced by ulcerations with consequent cicatricial contractions, by cancerous strictures, and by inflammatory (hypertrophic) conditions of the intestinal wall.

The ulcerations to be considered are the tubercular, stercoral, dysenteric, typhoid, and syphilitic. Tubercular ulcers are among the most frequent causes of intestinal stricture; the other forms have little practical importance. As is well known, syphilitic ulceration is most frequent in the lowest portions of the bowel, especially in the rectum, and is extremely rare in the upper portions of the intestine. Strictures due to previous ulcerative processes also occur in incarcerated herniæ and after traumatism. Finally, the peculiar strictures accompanying pernicious anæmia, to which Knud Faber[20] has recently called attention, must be mentioned.

Cancerous ulcerations are mainly found in the large bowel and rectum; they are characterized by a tendency to the formation of stricture. Since ulcerations, neoplasms, and their sequelæ have already been discussed in a separate section, a detailed account of these affections will not again be necessary.

Symptomatology and Diagnosis

As regards the intestinal stricture or obstruction, the symptomatology is the same as that described in the previous section. Special symptoms, when present, are evidenced by the special form of the underlying disease (tumour, syphilis, tuberculosis, dysentery, etc.).

This is true also of the *diagnosis*. Where constitutional changes or a characteristic tumour point directly to the cause of the affection, the correct diagnosis can be made; in other cases it may only be possible to say that there exists a stricture or total occlusion, or perhaps to determine approximately the portion of the bowel affected.

In stricture of the small intestine we should, on account of its frequency, first suspect tuberculosis as the probable cause. In stricture of the large intestine, exclusive of the cæcum, we must think of cancer. Syphilitic stenoses and stenoses due to sclerotic changes in the submucosa can not, in the present state of our knowledge, be positively diagnosticated.

VI. Obstruction from Foreign Bodies

In this category are included gallstones, intestinal concretions, instruments introduced per os or per anum, and inspissated fæces.

(*a*) *Obstruction by Gallstones*

This may occur in all parts of the intestinal canal from the pylorus to the rectum, but the different segments are not affected with equal frequency. Those most frequently involved are the lower portion of the ileum and the ileo-cæcal valve—i. e., the divisions which, for anatomical reasons (narrow lumen of the lower ileum, short taut mesentery), offer most resistance to the passage of large stones; next in frequency are the duodenum and jejunum. Obstruction by gallstones is rarer in the upper or middle portions of the ileum, and is extremely rare in the colon and rectum. In the vast majority of cases the stone passes into the intestine through a fistula which has resulted from inflammatory adhesions between the gall bladder and the intestines. Communication between gall bladder and duodenum is the most frequent occurrence, while that between small intestine and colon is rarer. Stones have been known to enter the duodenum through a choledocho-duodenal fistula. Gallstones may cause intestinal obstruction in other ways. Thus, Mikulicz[60] twice found gallstones, not in the intestinal canal, but in diverticuli of the cystic duct which lay across and compressed the duodenum. The observations of J. Israel[62] and Körte[62] have shown that smaller stones may produce intestinal obstruction, probably by exciting circular spastic contraction of the bowel. When a stone has been impacted for a long time, and is large and angular, it may produce inflammation and swelling of the intestinal wall, or even gangrene and peritonitis with or without perforation.

SYMPTOMATOLOGY

It is well known that obstruction by gallstones is met with more frequently in women than in men. Regarding age, Naunyn[57] has found among 120 cases only 5 under the age of thirty, and only 7 between thirty-one and forty years of age, while there were 96 cases between the ages of forty-one and sixty. After the latter period there is again a decided decrease in frequency.

In many cases (according to Lobstein[62] 17 times in 90) there is a previous history of attacks of biliary colic, or more rarely of jaundice. It is important to inquire whether the patient has suffered from paroxysmal attacks of so-called "stomach ache," which, as a rule, are nothing more than ill-defined attacks of cholelithiasis.

In other cases the history may point to a local peritonitis as the cause of the rupture into the intestines, or only a doubtful connection can be established.

The symptoms of intestinal obstruction vary widely according to the location of the stone. As already mentioned (page 346), the stone, when situated high up in the duodenum, produces symptoms of pyloric stenosis; in the descending portion of the duodenum continued bilious vomiting is one of the characteristic symptoms; still farther down, the usual symptoms of obstruction of the small intestines occur—reflex vomiting, which may very soon become feculent or even fæcal, visible or palpable intestinal peristalsis, more or less (generally less) meteorism, and finally retention of stool and gases. Naunyn[97], the greatest authority on cholelithiasis, observes that in intestinal obstruction by gallstones the retention of stool and flatus is not necessarily absolute. Collapse soon follows; it is seldom of the severe type met with in other forms of intestinal obstruction. In unfavourable cases death occurs between the fifth and tenth days of the disease, rarely later. There may be a favourable termination if the stone has been forced through the narrowest part of the gut—that is, if the stone has passed into the large intestine; here, however, it may remain for days before it is passed per anum. On the other hand, a cure does not result in all cases in which the stone has been passed. An intestinal lesion may remain which may later produce death from perforative peritonitis. Not rarely, as Naunyn[97] states, the lumen of the bowel may only temporarily remain pervious, and after days, or even weeks, again become obstructed. Finally, two attacks of obstruction by gallstones have been observed in the same individual. Reports show that if there be a fistulous communication between the gall bladder and colon, large stones may pass without causing symptoms of obstruction.

Diagnosis

In diagnosticating obstruction from gallstones it is necessary to determine that there really is a calculous obstruction, and, if possible, the site of the stone. In favourable cases (i. e., where the obstruction is situated high up) when evidences of former cholelithiasis or of a communication of the gall passages with the upper intestinal tract are present, it is possible to establish both the above facts. If the history is not reliable, and if objective signs of previous cholelithiasis (enlargement of the liver, painful gall bladder, and pressure sensitiveness of the posterior portion of the liver) are absent, a probable diagnosis may be made by exclusion. We must remember that incarceration, strangulation, volvulus, peritoneal adhesions, and internal stricture of the upper part of the intestinal canal (duodenum, jejunum) are relatively rare. Mistakes cannot, however, be avoided. If the stone is impacted lower down in the bowel, the diagnosis may be easy when a well-defined history is obtained, or when characteristic changes about the liver or gall bladder are found. In all cases of intestinal obstruction, therefore, one should carefully examine and palpate the liver and gall bladder.

Very rarely a tumour is palpable on the left or right side of the abdominal cavity (Kirmisson-Rochard[63], Sick[64], Köstlein[65], Dessauer[66]). Maclagan[67] has observed two cases in which a painful tumour was felt in the neighbourhood of the liver, and when this tumour disappeared symptoms of intestinal obstruction gradually developed. If none of the above-mentioned data can be obtained the differential diagnosis from other forms of intestinal obstruction will be difficult. When there is a suspicion of obstruction from gallstones repeated rectal and vaginal examinations should be made, for gallstones have thus been demonstrated in the intestines. Gallstones in the rectum may be digitally or instrumentally removed.

Visible intestinal peristalsis may make possible the localization of the obstruction, but it can seldom be observed, and is also found in other forms of intestinal obstruction.

As previously mentioned, the large intestine is rarely occluded by gallstones (1 case of Körte[61] and 2 cases of Courvoisier[68]). This may, however, occur in the rectum, when the peculiar symptoms (tenesmus, obstinate constipation, pain) will direct immediate attention to the site of the trouble.

(*b*) *Obstruction by Enteroliths*

We have already described the different varieties of intestinal concretions (page 112). They generally originate in the large intestine, for in this situation conditions are most favourable for the development of hard concretions. They are chiefly situated in the haustra coli or in the rectal ampulla. In the small intestine concretions due to stagnation of the contents may develop in the so-called true or false diverticuli. They occur most often in young persons, and particularly in the poorer classes who subsist mainly on vegetables, rather than in the better classes, whose diet contains more animal matter. Intestinal concretions form very gradually. "They may, moreover, be dormant, as it were, for years, or excite during that time but insignificant symptoms" (Treves).*

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* Loc. cit., S. 336 [and Intestinal Obstructions, page 199].

Symptomatology

The symptoms of this variety of obstruction resemble those of chronic stenosis, viz., constipation, attacks of vomiting, paroxysms of pain, and disturbances of general health. In a few cases large concretions have been felt through the abdominal walls or through the rectum. Occasionally fragments have been passed with the stools. By reason of its powerful muscular coat and its elasticity, the large intestine permits of the passage of very large concretions; hence severe symptoms of obstruction have very rarely been observed (case of Down[69]). Where the concretion is retained in the cæcum it may produce all the symptoms and sequelæ of typhlitis or perityphlitis.

Diagnosis

A positive diagnosis of an intestinal concretion can only be made when the concretion can be felt per rectum. The diagnosis is perhaps most likely to be made when a tumour is palpable, when symptoms of partial obstruction are present, when the general condition of the patient and the very protracted course of the affection speak against carcinoma, and when the customary food of the patient has been such as to favour the formation of concretions.

(c) *Obstruction from Entozoa* (*Ileus Verminosus*)

Opinion is still divided as to whether entozoa (chiefly ascarides) may cause intestinal obstruction. Leichtenstern[1], Davaine[70], and Heller[71] doubt its occurrence, while Mosler and Peiper[72] answer the question in the affirmative. In his Bibliographie d. klinischen Helminthologie, Huber cites 13 cases of obstruction caused by a large accumulation of ascarides in the intestines. It occurs almost exclusively in children. The ileo-cæcal valve is said to be the main site of the occlusion. Whether the obstruction is of a mechanical nature, or whether, as seems more probable, it is dynamic (reflex) in character, is as yet undecided. In the vast majority of cases the disease runs an unfavourable course. Heidenreich[73] has reported a successful case of enterostomy and Simon[74] one of colostomy for this condition. Very few of the observations heretofore made will stand critical examination; it is therefore impossible to describe the characteristic symptoms. The diagnosis is only possible through the accidental evacuation of ascarides, which is usually a result of therapeutic measures.

(*d*) *Obstruction by Foreign Bodies which have been Introduced*

Foreign bodies that have been purposely or accidentally introduced may reach the intestines through the mouth or anus. Most remarkable bodies have thus found their way into the intestinal canal. Neurotic patients have swallowed very dangerous articles, frequently with suicidal intent, but occasionally also when mentally deranged. Both old and recent literature is replete with such instances, which are simply medical curiosities and hence need only be mentioned. We refer, however, to the recent thorough article of Frikker[75], who describes a unique case which occurred in his own practice. Coins and similar articles, or false teeth, are most frequently swallowed.

SYMPTOMATOLOGY

If their size or length is not disproportionate to the lumen of the intestinal canal, swallowed articles generally pass through the bowels without trouble. This is also true of pointed or sharp bodies (nails, files, needles). In the majority of instances their expulsion is painless, and cause no symptoms of any kind.

If the foreign body is not passed, it is apt to be retained in certain portions of the alimentary canal — the stomach, lower segment of the ileum, duodenum, and more especially the cæcum and ampulla recti. Even then symptoms are not necessarily present; they may appear only after the expiration of months or years. There may be pain, colic, vomiting, more or less complete constipation, ulceration of the mucous membrane with perforative or local peritonitis, or with resultant stricture or abscess opening externally. After having passed through the intestinal wall, needles may be carried to different parts of the body, and extrude spontaneously or be artificially removed.

DIAGNOSIS

The diagnosis of swallowed foreign bodies can be made either from the history or from direct evidence. Regarding bodies introduced into the rectum direct evidence should by all means be obtained. In all other intestinal segments such evidence is only exceptionally obtainable by palpation. Metallic foreign bodies are best demonstrated and located by radiography.

My experience has shown that patients often imagine they have swallowed articles (needles, false teeth, etc.). I once "removed" a needle—presumably swallowed—from a hysterical female by showing her a needle in the water of an enema.

(e) *Obstruction from Fæcal Tumours*

In marked habitual constipation accumulations of fæcal matter may obstruct the lumen of the bowel. These tumours occur only in the large intestine, and particularly where the fæces are apt to be retarded—i. e., in the cæcum, at the flexures, and especially the sigmoid flexure. The longer the fæcal mass is retained, the greater is its loss in water and its chances for increase in size from further fæcal accumulation. Notwithstanding, these masses only exceptionally produce total obstruction, for there is always sufficient space for fluids and semisolid fæces to pass between the tumour and the intestinal wall.

Obstruction of the kind just described may produce other and occasionally serious consequences. The frequent development of sigmoid volvulus from impaction has already been mentioned. Furthermore, as described in the section on Cancer of the Large Intestine, a fæcal mass may be impacted in front of a stenosis in such a manner as to directly cause intestinal obstruction. As a result of stagnation of fæces, there may be a descent of intestinal coils, especially of the movable transverse colon, with consequent kinking or the formation of stercoral ulcers, and, in extreme cases, as a result of exhaustion of the muscular coat of the bowels, there may be complete arrest of the fæces. (See Ileus Paralyticus.)

SYMPTOMS

These are mainly those of chronic partial or complete intestinal obstruction. Some differences arise from the varying situations of the fæcal tumour. Thus, when situated low down (sigmoid flexure, rectum) there is tenesmus; when higher up the latter is absent. The most important symptom is the fæcal tumour itself, whose special peculiarities have been discussed in the General Division.

DIAGNOSIS

It is usually easy to recognise fæcal tumours in the lower portion of the large intestine by abdominal, rectal, or vaginal palpation. If the mass is situated in the upper portion of the large bowel, the diagnosis is more difficult, particularly when meteorism obscures the tumour. In such cases the history and clinical course alone, more especially, however, repeated observations of similar more or less severe attacks, may lead to the correct diagnosis. Even here, however, there must always be a lurking suspicion of some organic hindrance, of incomplete or beginning volvulus, peritoneal adhesions, kinkings, or compressions. It is still more difficult to decide whether the palpable fæcal tumour is the cause or result of the occlusion. Only complete, permanent relief from all the symptoms warrants a good prognosis.

VII. Obstruction without Physical Changes in the Intestines (Paralytic, Spastic, and Dynamic Obstructions)

Besides intestinal obstruction from mechanical hindrance, there is a second form in which the symptoms of obstruction are present without any discoverable cause. The older practitioners recognised this seemingly paradoxical condition. In 1865, Henrot, in a thesis classical even at the present time, gave an exhaustive description of these *pseudo-étranglements.*

The conditions are most readily understood when the intestines have been injured in some manner—e. g., by severe traumatism or continued constipation or abnormally great meteorism. At present we are unable to explain the origin of a sudden severe intestinal paresis. It may be theoretically explained, however, that there are marked changes in the innervating fibres of the muscular coat of the bowels, and that these changes cause the paresis. In other cases the paralysis is evidently due to reflex action (the *paralysie réflexe* of the French). A classic instance is that of the undescended testicle, which, when inflamed, may cause symptoms of intestinal obstruction; or, as recently demonstrated by numerous examples, an operation has been performed on the intestines, uterus, or ovaries, and there develops severe intestinal obstruction without any discoverable lesion in the intestinal wall itself. Whether the dynamic intestinal obstruction often found in peritonitis, and particularly in perityphlitis, is reflex, or whether, as Stokes maintained, it is caused by a serous infiltration of the bowel wall (collateral œdema), in which case modern writers would claim toxins as etiological factors, or finally, whether it is produced by lessened absorption (and hence accumulation) of gases, is a question which cannot at present be answered with certainty.

Based upon several observations, Leichtenstern[26] has proposed another theory to explain the intestinal paralysis of peritonitis.

According to this author, when the patient is lying on his back those distended intestinal coils which have long mesenteries are lifted up by the gases in them and pressed toward the anterior abdominal wall, while coils with fluid contents, particularly those with short mesenteries, cannot move or can move but little from the vertebral column. Because of their weight, the stagnant fluid contents of the duodenum or jejunum are much more likely to flow back into the stomach than they are to pass into the distended, paralytic bowel, which lies higher up and is held in place by the meteorism. This explanation, however, is sufficient only for isolated cases, since the above condition is absent in many cases of peritonitis with marked tympanitis.

Spastic obstruction (*ileus spasmodica*) is a second form of dynamic ileus, and one whose importance has only lately been acknowledged.

In spastic ileus there develops in any portion of the intestines a "spastic obstruction" which may be followed by the same changes as a mechanical obstruction. Heidenhain[76], by his vivisection experiments, has again brought this subject into prominence. Spastic obstruction occurs under the most widely different conditions: as primary spastic obstruction without discoverable cause, as a complication of hysteria (very remarkable instances have recently been described by von Leube[77] and Strauss), in gallstone obstruction without mechanical intestinal occlusion (J. Israel[42], Körte[61]), as a reflex condition in mechanical ileus (Heidenhain), in crises gastriques (Sandóz[78]), in tubercular intestinal ulcerations (Strehl[79]), and with foreign bodies in the rectum which do not entirely close its lumen (Grundzach[80]). The case described by the last-mentioned author appears to me to be an especially good example of spastic intestinal obstruction; I shall therefore briefly describe it:

After eating a hearty supper a man, thirty years of age, complained of abdominal pain, tenesmus, constipation, absence of flatus, and anorexia. Castor oil and calomel were given without result. Stomach and intestines markedly distended; abdomen sensitive to pressure; face anxious. Beginning intestinal obstruction was suspected. Rectal examination discovered a fishbone, 5 to 6 centimetres long, lying crosswise in the rectum. After extraction of the bone the patient very soon passed flatus and fluid stools.

Leichtenstern[1] is of the opinion that the intestinal spasm is simply a persistence of the muscle in a condition of elastic tension.

Nothnagel * also considers this possible. There are cases (for example, the one just described, and that of gallstone obstruction without occlusion) which point more conclusively to a spasmodic condition than to one due to intestinal paralysis. From a practical standpoint, however, both conditions are the same.

Symptomatology

The clinical picture of functional intestinal obstruction is so very similar to that of the various forms of mechanical obstruction already described, that we should only have to repeat what has been said. From this it will be seen that a demonstrable and objective differentiation between these two general forms is very difficult. It might be stated that visible peristalsis is absent in functional occlusion, but then it is also quite often absent in mechanical ileus.

Diagnosis

Under the above circumstances the diagnosis is extremely difficult. This is appreciable when, for example, we have to differentiate between peritonitis with intestinal paralysis and mechanical obstruction. In the introductory part of this chapter (page 368) we have described the factors that have to be considered. When there are symptoms of obstruction after operations upon the intestines or the sexual organs, or after reduction of a hernia, the conditions may be so favourable that a correct diagnosis is possible. Everybody who is acquainted with the literature of this subject knows that diagnostic errors are very apt to be made, for it will scarcely be possible to exclude apparently insignificant mechanical causes (slight adhesions, circumscribed peritonitis, intestinal compression, and crushing). For these reasons many surgeons are sceptical regarding dynamic obstruction.

Differential Diagnosis between the Several Forms of Intestinal Obstruction

In the majority of cases it is impossible to make a correct diagnosis of the variety and localization of the obstruction. In practice we must often be contented when we have approximately determined the site of the lesion and the probable cause of the occlusion.

The first question to be determined is whether the obstruction

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* Loc. cit., S. 360.

is in the large or the small intestine. In what follows, therefore, we shall briefly describe the symptoms which indicate the localization of the obstruction in one or the other parts of the intestinal tract.

(*a*) *Small Intestine.*—Here objective signs to a slight degree, but more especially the knowledge gained from experience, aid in making the diagnosis. Experience may be of great service. We herewith give an example: Sudden intestinal obstruction occurs in a young man who has always been well. No hernia; very rapid, extremely acute development, with vomiting soon becoming fæcal; no flatus or stool passed; intense indicanuria from the very onset of the disease; no marked meteorism. In such a case we may, without fear of error, diagnosticate obstruction of the small bowel. A second example is the following: An elderly woman, well except for obstinate habitual constipation, is attacked with symptoms of slight obstruction. No stool or flatus passed, slight vomiting, not fæcal in character; marked localized meteorism; marked tenesmus. Here we need have no great hesitation in diagnosticating an obstruction of the large bowel, probably a sigmoid volvulus.

No doubt there are cases in which from the very onset the symptoms are so obscure that the diagnosis of the site of the lesion is only a matter of personal experience, or rather of personal equation. Regarding such doubtful cases, it must be mentioned that occlusions of the large bowel generally present better *objective* signs than those of the small bowel. Rectal injections or inflation, recognition of a large, fixed, distended intestinal coil by means of auscultatory percussion, the possibility of palpating this intestinal coil, the previously described lumbar symptoms of Nothnagel (page 355); the recognition of mild peristalsis—all these combined will in favourable cases lead to comparatively certain conclusions. It has often been mentioned that examinations per rectum and vagina, and of the evacuations, may yield important, and, under certain circumstances, decisive results. Error is unavoidable in those apparently rare cases of simultaneous obstruction of both large and small intestines, described by Treves in his monograph, and lately, after several operations, by Hochenegg[81] (combination ileus). Furthermore, as already mentioned, the occurrence of multiple invaginations, cicatricial strictures, peritoneal adhesions, volvuli, etc., should be remembered. Such conditions cannot be recognised during life.

After the local diagnosis has been made other difficulties must be overcome. We can, however, often reach a diagnosis by exclusion.

The differential diagnosis of the nature of the obstruction must necessarily be limited to those conditions which are at all capable of being diagnosticated. Hence we must exclude constriction by omental bands, incarcerations by the appendix or by Meckel's diverticulum, by pseudo-membranes, fenestra or clefts in the mesentery, or by adhesions of several intestinal coils; occlusion by kinking or distortion, by the formation of knots, volvuli (excepting sigmoid volvulus); internal herniæ (with the possible exception of diaphragmatic hernia). We do not wish to imply that the diagnosis of these forms of obstruction can never be made, but that they can be recognised only under particularly favourable and rather accidental circumstances.

Deplorable as this is, it enables us to restore order out of this diagnostic chaos, by allowing us to study more carefully the few recognisable and distinguishable varieties of obstruction. For this purpose a detailed history, a careful study of the patient's condition, and an accurate knowledge of the symptomatology of intestinal obstruction are necessary.

As Graser[82] states, the history and the patient's daily condition should always be noted in writing, and any changes should be compared with previous observations. If careful observation at home is not possible, doubtful cases should immediately be sent to a suitable hospital.

Naunyn rightly observes that in all forms of intestinal obstruction the first practical question is whether or not an incarceration or a strangulation is present. In not a few instances this question may be answered affirmatively because of the severe initial symptoms—violent shock, almost constant intestinal pain, early vomiting soon becoming fæcal, and collapse very rapidly reaching the greatest possible severity. The important objective symptoms of incarceration and strangulation are the distended, fixed intestinal coil (occasionally with peristalsis), hemorrhagic exudate into the dependent portion of the abdomen, meteorism, moderate or absent, large intestinal hemorrhages (rare) (Naunyn), and marked indicanuria from the beginning of the disease. The first symptom (von Wahl's) is the most important, but unfortunately it is not always well marked. As previously mentioned, it is entirely absent when the strangulation affects a large part of the intestine. In such cases it is impossible to diagnosticate more than the site of obstruction. Nothnagel's case (mentioned on page 382) shows that there are exceptions even to these rules.

Among the forms of obstruction of the small intestine that can be diagnosticated we must consider obstruction by gallstones. The facts that are of value for the diagnosis of doubtful cases—and these alone are here considered—are the age of the patient (generally over thirty-one years), sex (more frequent in the female), the history (which may, however, leave us entirely in the dark), some change (though but slightly indicated) in the liver, especially the protracted course, the passing of flatus, and, despite fæcal vomiting, of stool, and, finally, the slight tympanites. Hemorrhages per anum do not speak against gallstone obstruction. When the obstruction is situated high up—i. e., in the vicinity of the papilla—the picture is in itself very characteristic, although the cause of the occlusion may remain obscure if the history does not contain data which call attention to the possibility of intestinal obstruction by a gallstone. The case might, however, be one of obstruction due to compression by neighbouring tumours (which need not necessarily be palpable), by adhesions with surrounding tissues (gall bladder, pylorus, mesentery, etc.), by ulcers with cicatrization (Varr), and by torsion of the mesentery (J. Schnitzler).

Foreign bodies impacted in the small bowel can be diagnosticated only when there is a history of their having been introduced; otherwise this is not distinguishable from other forms of obstruction, especially that by gallstones.

(*b*) As already mentioned, *obstructions of the large intestine* are more readily recognised and their nature determined than are obstructions of the small intestine. There is less danger of an error in intussusception than in any other form. Its occurrence in very young children, its sudden onset, the severe but generally intermittent pain, the tenesmus, the characteristic discharges (especially the admixture of pus and blood), the presence of a smooth, growing, and wandering tumour which occasionally becomes tetanically contracted and is sometimes directly palpable and visible per rectum, in some cases the passing of a sloughing, gangrenous intussusception—all these symptoms, taken together or separately, are so characteristic that doubt as to the correct diagnosis can occur only under particularly unfavourable circumstances. In the differential diagnosis we must consider rectal polypi with bloody stools (which are, however, easily distinguished from invaginations), acute dysentery, and severe intestinal colic. In the early stage of the disease it is sometimes impossible to distinguish the latter condition from intussusception, but the diagnosis is almost always cleared up by the further course of the affection. On the other hand, it is usually impossible to correctly diagnosticate an invagination accompanied by a neoplasm.

Generally, volvulus of the sigmoid, the second most frequent form of obstruction of the large bowel, also presents a characteristic picture. Its main diagnostic features are the occurrence late in life and in persons suffering from obstinate constipation, the sudden development combined with a relatively slow course, absence of fæcal vomiting despite complete intestinal obstruction, futility of rectal injections of large quantities of water* (less than one half litre), marked meteorism, which may extend over the whole abdomen or may become quite visible and be limited to a fixed coil, but without peristalsis, frequent tenesmus, and finally slight indicanuria. Bloody evacuations occur in volvulus of the sigmoid flexure as well as of the small intestine, in invaginations, and but rarely in obstructions from gallstones. The other symptoms vary so widely that only rarely ought there to be any difficulty in differentiating between these conditions. We may, however, have to determine whether we are dealing with a simple volvulus, or with a volvulus produced by a carcinomatous stricture or a tumour. When a tumour is absent, it will generally be impossible to make the differentiation. In itself the clinical picture is not sufficiently characteristic to enable us to state whether we are dealing with a complete volvulus of 360 degrees or with one of 180 degrees.

Occlusions of the large intestine which develop in the later stages of a stricture (generally carcinomatous or tubercular) are also easily recognisable. If a tumour is present, there can only be doubt as to its nature; frequently the further course of the disease will clear up the case (compare chapter on Intestinal Neoplasms). If there is no tumour, we can arrive at more than a probable diagnosis only in the presence of other definite clinical data—tubercular symptoms, pyrexia, age, metastases, cancer cachexia, etc. Usually it is impossible to determine whether the obstruction is caused by paralysis of the intestinal wall, or by foreign bodies or fæces impacted in front of the stenosis, or by other mechanical factors.

Foreign bodies in the large intestine which have been swallowed can be diagnosticated from the history; others can only be recognised when found in the rectum, or when fragments of them are accidentally passed per rectum. Of importance, too, is the fact that enteroliths are generally found in the cæcum, and produce symptoms of typhlitis or intestinal stenosis before the symptoms of obstruction appear. Occasionally a tumour may be felt in the cæcum, but its nature will be doubtful. On the whole, however, enteroliths will seldom have to be considered in the differential diagnosis.

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* Compare, however, limitations, given on page 382.

As mentioned in the general division (p. 76), *fæcal tumours* of the large bowel have often given rise to error. One who has had any experience with these pseudo-tumours will, I believe, scarcely ever be misled. Even when these tumours consist of old residua, their compressibility and elasticity will usually indicate the proper diagnosis. Difficulty will only arise when, in addition to the fæcal tumour, a real neoplasm is present, or when the clinical course points to a neoplasm. The case taken from Nothnagel's Diseases of the Intestines, and cited on page 77, is a very good example of this. For the diagnosis of a fæcal tumour, the clinical course is very important, and the history may give us valuable information. Fæcal tumours produce symptoms of occlusion very gradually, and these, when present, are relatively mild. The general health is but little affected; there is absence of fæcal vomiting, of a fixed distended intestinal coil (a point of differentiation from volvulus of the large intestine), and of visible peristalsis. This latter condition is always present in stricture of the large bowel.

(*c*) *Dynamic Intestinal Obstruction.*—If we obtain a history or other evidence of laparotomies or operations on the female genitals, of traumatism, reduced herniæ, the diagnosis of functional (dynamic) obstruction ought not to be very difficult; but even in these cases a positive differentiation is sometimes not easy. For example, in obstruction following hernial reduction it is hard to state whether the obstruction is of a mechanical (incomplete reduction, adhesions of incarcerated intestinal coils) or of a functional nature; or, when a volvulus has been untwisted, it is difficult to state whether the intestine has not been twisted anew, or whether the volvulus was the only obstructing factor; or whether we are not dealing with an obstruction of a dynamic type. As will be seen when we shall come to speak of the treatment, these questions are not purely hypothetical. It is important for the physician to know them before he begins treatment. The differential diagnosis can be made only by a study of each separate case and by the most careful consideration of all details. When applied to a complicated case, every tabulated scheme utterly fails.

We have already mentioned that in these cases we must also think of hysteria, for the latter condition may cause much diagnostic difficulty.

Since it has been thoroughly discussed (p. 368, etc.), we shall not enter into the differentiation between mechanical and dynamic ileus and acute peritonitis.

It is evident that the varieties of obstruction which permit of a positive diagnosis are not numerous, and even this small group presents new phases and abnormalities which may absolutely disguise the typical clinical *ensemble*.

THE TREATMENT OF STRICTURES AND OBSTRUCTIONS OF THE INTESTINE

Although physicians differ in many points regarding the treatment of these affections, all agree that we are concerned with one of the most difficult and responsible fields of internal pathology. This is owing, no doubt, to the uncertainty which attends all cases which can be recognised clinically, but more especially those far more numerous cases in which the diagnosis is obscure. Even in so simple a condition as internal stenosis of the large bowel we cannot be certain of the degree of obstruction or the condition of the muscular layers of the gut above the seat of obstruction. We can note the efforts of the hypertrophied intestinal segment to force fæcal matter through the stricture, but who can say whether it will finally succeed, and, if so, when? What is the anatomical condition of the stricture? How far has it advanced peripherally? Is rupture impending? It is almost impossible to answer any of these questions. This is also true of intestinal occlusions. Let us take the simplest example, that of volvulus of the sigmoid flexure. Is the volvulus complete, or incomplete? Is it primary, or caused by a cicatricial stricture of benign or malignant character? What is the condition of the twisted bowel? Is there danger of peritonitis? Is it beginning, or has it already begun? These questions are of the greatest importance both as regards the treatment to be instituted and the life of the patient; still it is impossible to clearly and precisely answer any of them. If, then, typical cases are so perplexing, how much greater the difficulty in cases which do not even admit of localization, let alone of an anatomical diagnosis.

The difficulty in the selection of a method of treatment is due not so much to the doubtfulness of the gross diagnosis, as to the fact that we know so little of the condition of the occluded segment during any stage of the disease. Other considerations influence our action in individual cases—viz., the age of the patient, history of previous exhausting disease, general condition, complicating constitutional dyscrasiæ, etc. Owing to these difficulties, it is impossible to give a schematic account of the treatment of intestinal obstruction. The efforts of both surgeons and medical men in different countries have hitherto been unsatisfactory, because they have had no common facts on which to base their plans of treatment. Only a most comprehensive collective study, such as has been so well carried on by Sahli in perityphlitis, may clear up the disputed points. At present we must be content to give an account of what can be gathered from the rich surgical and medical literature of the subject, and from my own experience concerning the different methods of treatment.

I. Treatment of Intestinal Strictures

Unless we are dealing with a foreign body which may be passed *per vias naturales*, it is impossible to cure an intestinal stricture by internal medication. The medical practitioner can only assist in the efforts already begun by nature to compensate for the obstruction. We possess several means to attain this end: appropriate diet and mechanical and medical remedies.

(*a*) *Diet.*—In the General Division we have described the appropriate nourishment in intestinal stenosis. In the section on Intestinal Carcinoma (page 323) there are special comments which may be directly applied to the other forms of chronic stricture of the bowel.

While referring the reader to the above sections, we shall here briefly recapitulate the most important facts. The caloric value of the diet must be as great as possible. The lower the site of the stricture the less will be the difficulty of carrying out these principles. The higher the occlusion, the greater will be the accumulation of undigested material. The condition is similar to that in gastrectasia; the same difficulties are encountered in stenoses of the small bowel situated high up as in dilatation of the stomach.

We must again call attention to the necessity of avoiding food indigestible or difficult of digestion, especially if it contain much cellulose.

In stenosis of the small intestine the food should be fluid or semifluid, and should contain appropriate physiological laxatives. We need not regard the general warning against so-called flatulent food, which exists only in the imaginations of thoughtless people; we should outline the diet according to the individual case. The practitioner who distributes printed diet sheets shows that he has no conception of the importance of diet in modern therapy. In far advanced stenosis of the small intestine nutrient enemata may be required. For the details or technic of this procedure the reader is referred to other works on this subject.*

(*b*) *Mechanical Treatment.*—This consists in the use of stomach lavage and rectal enemata. The former is used only in stenoses of the small intestine. Since its technic is quite similar to that employed in dilatation of the stomach, we refer the reader to textbooks on diseases of that organ. Rectal enemata may be employed in stenosis of both the large and the small intestines when, as frequently happens, laxatives must be avoided. They may be of great value in stenosis of the small intestine, and in high stenoses of the large intestine (occasionally in connection with mild laxatives). Thorough irrigation of the rectum is most appropriate, or else small enemata of oil or soap, with glycerin, oil, cod liver oil, etc., may be given. (Regarding particulars, see Chapter XI.)

(*c*) *Medical Treatment.*—The first question to be considered is the treatment by laxatives. They are indispensable in the therapy of intestinal stenosis, for, with but few exceptions, the patients suffer from constipation or from alternating constipation and diarrhœa. We have discussed this subject in the General Division (page 189), but must again emphasize that drastic purges are always contraindicated.

Opium and its alkaloids, as well as the belladonna preparations, are used in intestinal stenosis either as sedatives or (and this cannot be too strongly emphasized) as laxatives. This is especially true where there is visible and palpable intestinal spasm. Thus, in a case of stenosing cancer of the cæcum which I had observed for a long time, and in which symptoms of acute occlusion were impending, the use of a suppository of opium and belladonna was followed by a daily well-formed stool.

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* See Boas, Diagnostik u. Therapie d. Magenkrankheiten, 4te Aufl., 1897, Th. 1, S. 293; and von Leube, in von Leyden's Handbuch der Ernährungstherapie, 1897, Bd. i, S. 490.

Surgical Treatment of Intestinal Strictures

As already remarked, internal medication cannot have a curative effect upon an intestinal stenosis unless the same be due to a foreign body. In every case of intestinal stenosis we must therefore consider the advisability of operation. The question of the cause of the stenosis will naturally play a very important part as an indication. A malignant tumour, no matter where situated, offers a worse prognosis than a cicatricial stenosis or a stricture produced by a foreign body—for instance, by gallstones. The site of the stenosis must also be considered. Owing to the immobility of the duodenum, a carcinoma of its superior portion can scarcely be radically extirpated, while tumours of the ileum or of the colon offer much better chances of complete removal. Benign tumours, though of large size, are generally more amenable to radical treatment than malignant ones. Extensive adhesions may make the removal of the tumour much more difficult, and after its removal may cause intestinal obstruction. Autopsy often reveals multiple strictures, whereas at the time of operation but one stricture was found and removed (Hofmeister and others). It is never possible therefore to accurately determine beforehand the nature of the operation required, or its result and the prognosis.

The indications for operation vary with the individual case. Generally speaking, benign stenoses, unaccompanied by marked mechanical disturbances, are best treated by internal methods, while severe stenoses, in which internal measures have been exhausted, are proper subjects for surgical treatment. Mild stenoses of a malignant nature (tuberculous and carcinomatous) belong exclusively to surgery. In these cases delay may be fatal. On the other hand, we have seen that the diagnosis at this stage of the disease is extremely difficult. Patients with a beginning stenosis are very rarely willing to be operated on. When complete obstruction supervenes upon stenosis the treatment is in every respect the same as that described under that affection.

II. Treatment of Intestinal Obstruction

When the practitioner is confronted with a case of intestinal obstruction he ought to have a clear idea of the limitations in our knowledge of this most dangerous form of intestinal disease. It would be of incalculable value for therapeusis if by some method similar to radiography we could actually see how an intestinal segment is caught in a mesenteric fenestrum, or how a peritoneal strand causes intestinal strangulation, or how an angular kinking of the bowel is produced by a cheesy degenerated gland, or how a foreign body has obstructed the intestinal lumen. We could then immediately exclude a number of cases in which internal treatment is useless. These are the mechanical obstructions, most of which can only be removed by mechanical means.

Since our present knowledge does not enable us to localize or to recognise the cause of the obstruction in all cases, it is difficult to lay down absolute rules for the physician's guidance. Is internal treatment absolutely of no avail from the very beginning of the case, or shall we wait? How long shall we wait? Or is it a case in which we can expect so much from internal treatment that surgical treatment is uncalled for? In most cases it is scarcely possible to answer these questions, and therefore our treatment must be somewhat schematic. We would not have it inferred that all cases of obstruction are to be treated alike. On the contrary, we should try to individualize as much as possible.

Internal Treatment of Intestinal Obstructions

This may be divided into dietetic, mechanical, and medical measures.

(*a*) *Diet.*—Most clinicians agree that patients with intestinal obstruction should have an "absolute diet" [withdrawal of all food] (Goltdammer, Curschmann, Ewald, Nothnagel, etc.). I have already expressed myself (page 152) as somewhat averse to this opinion, and I would again explain my views on this subject.* In my opinion the diet in obstruction of the large intestine must be quite different from that in obstruction of the small bowel. As Curschmann[43] states, feeding *per os* in the latter disease really imposes an additional burden on the intestine, and hence should be limited as much as possible, if not entirely given up. It is different, however, in obstruction of the large intestine. In these vomiting does not always occur; when present it is not usually very violent, and, what is very important, it very rarely becomes fæcal. The view often expressed, that there is a suspension of the motor and of the resorptive power of the intestinal segment lying proximal to the obstruction, is true of the small intestine, but as regards the large intestine is, so far as I can see, only a pure hypothesis, and has never been experimentally proved. Accordingly, it is entirely proper to have patients with obstruction of the large bowel consume as much easily digested nourishment as possible. We must proceed carefully and cautiously, and be led by the course of the affection in each individual patient.

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* Above all, we must discard the idea that diet is of secondary importance in intestinal obstruction. I believe that patients have lost their lives in consequence of this misconception, which is directly opposed to modern views. I consider everything of caloric value consumed by the patient as so much gain for him.

The nourishment must be regulated even in its smallest detail. It should be a fluid or a semifluid diet, which is absorbed without difficulty in the upper bowel and leaves no residue. The following are particularly to be recommended: Iced milk, beef tea, caudle, meat jellies, and the numerous albuminoid preparations found in the market. We may also try small quantities of alcohol in the form of cognac, Hungarian wine, sherry, etc. It is not a question of a great nutritive result, but when we consider that during the course of treatment an operation is often indicated, we must never even for an instant forget the importance of stimulation of the heart, and of giving the patient large quantities of fluid.

When from the onset, or from attempts already made, we know that patients cannot be fed by the mouth, we should always employ nutrient rectal enemata. Where for mechanical causes the enemata are not retained, we may (as recommended by Curschmann) attempt to obviate the threatening systemic loss of water and the cardiac collapse by subcutaneous injections of salt and sugar solutions. Many surgeons appreciate the value of the latter procedure, and before beginning operations on patients with cardiac weakness are in the habit of injecting saline solutions.

(*b*) *Mechanical Treatment.*—*Gastric lavage*, introduced into the therapy of intestinal obstruction by Kussmaul and Cohn, undoubtedly is the most valuable of our mechanical measures. Its effect is to lessen the pressure above the occluded segment and thereby to remove the great hindrance to compensation of the obstruction. We have already described the value, indications, and technic of stomach lavage (p. 184), and we would here again point out that this procedure is indicated only in cases of obstruction of the small bowel. Gastric lavage will scarcely ever be attended by any great success in invaginations of the large bowel, in sigmoid volvulus, in internal strictures of the large bowel, in dynamic ileus, in obstruction by impacted fæces or enteroliths.

Under certain conditions *rectal injections* may favourably influence the course of the intestinal obstruction, only, however, when the latter is deeply situated. Invaginations may be relieved, impacted foreign bodies loosened, and incomplete volvulus occasionally reduced, inspissated fæcal masses gradually softened, and the paralytic large intestine excited to peristalsis. I consider thorough rectal irrigations with soap or emulsified oil solutions (see p. 179) most appropriate. We must naturally not expect too much from these procedures.

A few observers have recommended *massage* in intestinal obstruction. Except perhaps in positive obstruction [!] its use is not sufficiently clear. Because of the danger of peritonitis, we would further recommend the greatest caution in such manipulations.

Regarding puncture of the distended intestine by the Pravaz syringe, opinions are very much at variance. This procedure has lately been recommended particularly by Curschmann[83], O. Rosenbach[84], Fürbringer[85], von Ziemssen[86], and others. On the other hand, it has been condemned by almost all surgeons; for example, by Treves,* Körte[87], Graser,† Kocher[88], and others. Kocher calls it an operation in the dark.

Curschmann[83] recommends the following technic for puncture of the bowel through the abdominal wall: A long needle, having a valve of the calibre of a Pravaz aspirator, is thoroughly disinfected. After the valve has been closed the needle is passed into the intestinal coil which is most distended, and which in each case must be most carefully sought for. The needle is then immediately connected with a rubber tube, the latter is passed into a bottle containing a watery solution of salicylic acid, and the bottle is turned over a basin containing the same fluid. When the valve of the canula is opened the intestinal gases rise into the bottle, at first in a continuous stream, later more slowly in large pearls, finally intermittently. Fürbringer's advice not to hold the needle tightly, but to allow its direction to be guided by the intestine itself, is important. The intestines may be punctured at various places.

This method has for its object the diminution of the tension in one or more intestinal coils, thereby allowing the incarcerated segment to free itself more readily. Curschmann and Fürbringer claim to have seen cures from the procedure. The method requires great caution and should only be used in selected cases. It is only indicated in cases with well-defined distended coils, most frequently in strangulation and volvulus. It is strongly contraindicated in intestinal paralysis, in invaginations, in peritoneal irritation, especially when a tendency to gangrene exists.

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* Darmobstruction, S. 446 u. f. [Intestinal Obstructions, p. 471 et seq.].

† Loc. cit., p. 602.

Inflation with air has practically the same significance as enemata of water. Many cures by this method, particularly in invaginations, have been published. Impacted foreign bodies may also be removed in this manner. Finally, inflation with air may temporarily or permanently relieve intestinal kinking. In intestinal ulceration and peritonitis, however, it is strongly contraindicated.

Electrical Treatment.—By means of the rectal sounds already described (page 178) the faradic or galvanic current may be used for the relief of obstruction. Both currents have been employed with marked success. Very favourable results were obtained by Bodet, of Paris, who reported 53 cures in 70 cases (!). In France electrical treatment is considered of extraordinary value, and is preferred to opium. Boudet[89] uses the galvanic current exclusively. After the rectum has been filled with one litre of salt solution, the current is applied by means of a soft-rubber sound, which in its interior contains a metallic wire. The instrument is similar to the rectal sound described on page 174. The negative pole is connected with the sound, and the positive pole is applied to the abdomen or back by means of a broad flat plate electrode. The current applied varies from 10 to 50 milliampères, and each application should last from 20 to 25 minutes. We would naturally hesitate to ascribe to the electric current such marked effects in the removal of mechanical obstructions, but the high percentage of the reported cures refutes the objection that *all* these were cases of fæcal impaction. This method deserves more consideration in Germany than it has heretofore received.

(*c*) *Medicinal Treatment.*—Practically this is limited to either laxatives or sedatives. The relative value of these two classes of remedies has been in dispute for more than a century, and only within the last ten years has the conclusion been reached that laxatives are contraindicated in all forms of obstruction except fæcal impaction and paralytic obstruction. Heidenhain[90] has recently pointed out that after hernial reduction and operations for intestinal obstruction the only proper treatment consists in cleansing enemata or laxatives, particularly castor oil. In this we fully agree with him.

In all other cases treatment by laxatives can only do harm. In a case of chronic invagination reported from Kussmaul's clinic[91], a dose of castor oil was followed by marked abdominal pain and by rigidity of the intussusception, which descended to the anus. After morphin, on the contrary, the pains not only decreased, but the tumour became less marked and the invaginated portion retracted. When we consider that during the first stage of occlusion there is an attempt to overcome the presenting obstacle by increased peristalsis, it is evident that an artificial increase of these efforts is entirely unnecessary and generally harmful. The further filling of the distended intestine above the obstruction lessens the chances of spontaneous cure.

With the exception of the two above-named instances, opiates spulvus opii, 0.02 to 0.05 grams every three or four hours, or morphin, 0.01 to 0.03 [!] grams) act effectually. They quiet pain, control violent peristalsis, prevent initial shock, diminish vomiting, induce sleep, and thus favourably influence the general health. Undoubtedly opium, similar to digitalis, is a tonic in heart failure. By quieting the intestines, and preventing dragging upon the inflamed peritoneum, opium acts as a prophylactic against local peritonitis, which, according to present views, may develop in the first stages of obstruction. Opium is best administered in the form of suppositories (each containing 0.05 grams), or subcutaneously ([extr. opii] 0.01 to 0.05 grams). I would recommend the latter method. This treatment, however, has its indications and its limitations. I repeat that opium is to be used only in the first stages of intestinal obstruction, and not when general weakness or cardiac paralysis has appeared. The latter instances may be those in which, as some surgeons believe, the narcotic temporarily masks the serious symptoms of obstruction. This justifies the advice given by some authors, that after the therapeutic effect of opium has been obtained one should stop its administration for a short time, so that the actual condition may not be obscured by any medicine. Opium is also strongly indicated when the occlusion is complicated by local or general peritonitis. Though most of these latter cases die, opium therapy at least presents the *possibility* of limiting the inflammation.

Thus we possess many remedies and methods for the internal treatment of obstruction. So far as I can see, all are agreed upon the means to be used, but not upon the manner of their administration. Some advise using all measures at one time, others that they be used in rapid succession. I believe everything depends upon the diagnosis. For example, gastric lavage is not applicable to sigmoid volvulus or invagination; here one should give air insufflations or water enemata. Furthermore, we should advise appropriate diet, methodical opium treatment, and always use the constant electrical current. In strangulation of the small bowel we should place our main reliance upon opium and gastric lavage. The cardiac power may be increased by small rectal enemata, injections of saline solution, etc. We should make it our duty to use each remedy systematically and persistently, and not discard one method before its favourable or unfavourable results have been demonstrated. Misdirected overtherapeusis may do more harm than carefully planned expectant treatment.

Surgical Treatment of Intestinal Obstruction

No surgeon, however experienced, can in every case of intestinal obstruction state positively whether, and at what moment, an operation may be necessary. Even if his judgment in one or more instances was correct, the very next case might demonstrate that this judgment was only accidental.

A general rule of procedure must be the result of principles derived from many observations; but even then it is almost impossible to formulate satisfactory principles. This is due, above all, to the variability of the material upon which the private physician, the hospital physician, and the surgeon must base their judgment. In the majority of instances the surgeon sees only the severest cases of intestinal obstruction and but few of those which have been cured medicinally, while the hospital physician, and more especially the private physician, frequently succeed in curing cases by conservative treatment. In view of the large number of cures (33 to 35 per cent, according to fairly well agreed statistics), and of the rarity of obstruction from fæcal impaction, the objection advanced by some surgeons that these are only cases of simple impaction does not come into serious consideration. In which forms of obstruction do medical cures occur? They undoubtedly include cases of fæcal tumour, gallstone obstruction, invagination, and, as Curschman[83] has shown by autopsies, kinking of both limbs of a jejunal coil and volvulus of the sigmoid. *This proves nothing more or less than the possibility of curing even severe forms of intestinal obstruction by internal therapeutics* (*not, however, as some surgeons claim, by the opium therapeusis*). Thus the extreme view of a few surgeons, that every case of mechanical obstruction requires operation, lacks confirmation.

It is certain, however, that operative procedures have saved many lives which would otherwise have been lost. From statistics of 288 cases, Naunyn[37] has demonstrated that the earlier the operation the better the results obtained. Of those operated during the first two days of the disease, 75 per cent, and of those operated after the third day, 35 to 40 per cent, were cured. These statistics do not refer to all cases of obstruction, but only to those which were operated upon. They serve to emphasize the statement that if operation is to be performed at all it should be done as early as possible.

Since the results of internal treatment, though not exactly favourable, are by no means hopeless, they may be compared with those following operation. If we consider only the statistics of individual prominent surgeons, we shall find that the results of surgical interference are not particularly encouraging. Thus, in about 110 cases of intestinal obstruction, Obalinski[36] had a mortality of 34.5 per cent—just as large a death rate as those cases treated internally. Treves,* who reported 122 cases of laparotomy up to the year 1888, had about the same mortality, 36.9 per cent. Even Kocher[88], whose results in abdominal surgery are equal to any in the world, had as high a mortality as 38 per cent. Hence we may conclude that improved antisepsis and technic have not improved the unfavourable results of operation. We must therefore search for another factor. In his discussion on intestinal obstruction (1889) Schede has spoken so convincingly that I cannot but reproduce his own remarks: "Every surgeon, particularly every abdominal surgeon, must agree with me that these operations [for obstruction], if performed upon healthy individuals, would only very rarely end fatally —in 5 per cent, or at most 10 per cent, of the cases. But there is scarcely any other condition which so rapidly lessens and so severely taxes the ability of a patient to withstand large operations and long abdominal manipulations as intestinal obstruction. A few days are often sufficient to bring about a condition in which the much-weakened patient is unable to bear the simplest operation—for example, the search for and division of a pseudo ligament."

In his recent treatise Kocher has sought for the conditions which produce these unfavourable results. He found them in the changes which the intestine suffers in consequence of disturbances in its circulation. The epithelium of the obstructed segment is destroyed, allowing micro-organisms to find their way into the peritoneum, and cause peritonitis. Putrefying substances are more easily absorbed from a mucous membrane denuded of its epithelium, hence an auto-intoxication (or sepsis) results. Kocher has likewise called attention to the danger of perforation from ulcers which develop above the stricture ("distention ulcers").

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* Darmobstruction, S. 461.

The chances of operative success are also considerably lessened by the difficulty, even during operation, in recognising existing conditions. Any one who has witnessed the efforts and the time required to control and replace the distended intestines which continually protrude from the abdominal cavity will understand that the strength of the patient is often exhausted before the actual operation begins.

In addition, it is often impossible before operation to determine the site or the type of obstruction.

Where shall the incision be made? Which intestinal segment shall be sought for? Is there one obstruction, or, as occasionally happens, are there several, and, in the latter instance, which one occludes the bowel? From this it can be readily understood that the surgeon is often confronted with insurmountable difficulties.

If we are to draw the proper conclusions from this discussion, we must concede that the results and dangers of internal and operative treatment are about the same. In any given case, therefore, it will be the duty of the physician to carefully consider which method —operative or internal treatment—offers the better chances for recovery.

The special conditions of the case, the kind of obstruction, the age and strength of the patient, and the results obtained by internal therapy, are factors to be considered. The sum total of experiences reported, favourable as well as unfavourable, must also influence us in our decision. Regarding this point the following statements may be made:

In the most severe forms of obstruction—*strangulation and incarceration*—the chances of cure by internal measures are very small indeed. If in the very beginning of the disease the symptoms are severe and the clinical signs of internal strangulation are present, and if a short trial with opiates and gastric lavage has been without result, operative interference is undoubtedly indicated.

In those cases of obstruction due to an old or recent external hernia, the indication for operation is also very clear. Statistics show that here the chances of cure are much better than in any other form of obstruction. According to Naunyn, 72 per cent of the cases are cured. These favourable results have been obtained because the diagnosis can frequently be made before operation, and the field of operation is limited.

In gallstone obstruction the results are not so satisfactory, and we must be more guarded in our prognosis. The chances of spontaneous cure are comparatively good (according to Courvoisier, 56 per cent, Lobstein, 52 per cent, Naunyn-Schüler and Dufort, 44 per cent), while for the operated cases Naunyn (23 cases) reports a mortality of 70 per cent, and Lobstein (33 cases) a mortality of 60.1 per cent.

From this it follows that surgical treatment of gallstone obstruction does not as yet offer such favourable results that operation ought be immediately advised. Since the symptoms of this form of obstruction generally develop slowly, we should first carefully try the internal therapeutic measures which have been described. The time for operation must be decided upon in each individual case.

Regarding operation for other foreign bodies there are not at present sufficiently extensive statistics from which to draw conclusions. It is a well-known fact that even large bodies may pass through the intestines without difficulty. Since in the majority of these cases operation is followed by favourable results, we should not too long employ internal therapeutic measures if intestinal obstruction develops. Obstruction from ascarides (a rare form) may also demand operation.

Most authorities agree that as soon as the diagnosis of invagination is made the case should be handed over to the surgeon. Statistics bear out the complaint of surgeons that patients with invagination are generally referred to them too late. Barker[92], for example, cured 7 out of 11 cases, a result which he declares is due to his operating as early as possible. According to Gibson[93], who in his article gave a table of all cases (239) operated on up to the year 1896, the general mortality is 53 per cent; in those operated on on the first and second days the mortality is only 39 to 41 per cent; on the third day, 62 per cent; on the fourth and fifth days, 72 per cent; and on the sixth day, 100 per cent. He explains these differences by the fact that even as early as the second day 14 per cent of the cases are already irreducible; on the third day, 38 per cent; on the fourth, 57 per cent; and on the seventh, 80 per cent. Gibson gives the proportion of deaths in the reducible and irreducible invaginations as 38 : 82. The age of the patient naturally plays a great part in the indication for operation. Gibson's statistics show a mortality of 82 per cent in patients less than three months old; the mortality gradually decreases up to the tenth year, when it is 37 per cent, increasing again to the fifteenth year (68 per cent); after this it approaches the adult mortality (62 per cent).

These figures show that, even in cases operated on early, the death rate from invagination is considerable. The results of conservative treatment are still more unfavourable.

Since the above statistics demonstrate that early operations (up to the third day) offer the most favourable chances, we should not continue too long with palliative treatment. Surgeons state, however, that palliative treatment should not be entirely neglected.

For example, Rydygier[94], in his latest work, recommends non-surgical treatment at first—electricity, gastric lavage, rectal enemata and distention with gas in the knee-chest position, massage, and attempts at reduction of the invagination in deep narcosis. Only when one or all of these measures fail is operation to be resorted to.

Chronic invagination is somewhat more amenable to conservative treatment, and the appropriate therapeutic measures may for some time be tried. If these are ineffectual and the patient's condition becomes alarming, it is best to operate at once. According to Rydygier's rather small statistics, the mortality from operation is only 24 per cent.

In volvulus, particularly of the sigmoid, the indications for operation are different. Naunyn[87] advises delay and individual consideration of the cases. The results of surgical treatment are by no means excellent, though in 19 cases of volvulus of the sigmoid (in 14 of which the correct diagnosis was made) Obalinski cured 10 by operation. Nothnagel * does not agree with Naunyn's conclusions, and pleads for active interference not alone in severe but also in mild cases. For the following reasons I am inclined to agree with Nothnagel. It is not particularly difficult to diagnosticate sigmoid volvulus, the surgeon can readily find the obstruction, and, if the volvulus was not originally incomplete, the chances of spontaneous reduction are very small. By manipulations through the rectum, we shall only rarely succeed in producing a permanent cure. On the other hand, we must remember that the symptoms of volvulus usually develop slowly, that there is usually no fæcal vomiting, and that the patient's general condition remains satisfactory for quite a time. Accordingly, unless the disease assume an exceptionally severe course, we may wait two or three days for a spontaneous reduction. If within this time this has not occurred, operative

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* Darmkrankheiten, S. 425.

interference is at once necessary. If, as is very often the case, a carcinoma or other tumour is the underlying cause of the volvulus, there will be all the more reason for surgical interference, since by operation we not only relieve the volvulus, but also may remove its cause. A spontaneous reduction will not permanently cure the condition, as the volvulus is very apt to recur.

All authorities agree that the surgical is the only proper treatment for *internal intestinal strictures.* Since these generally develop slowly, the question of operation will have to be decided before there is complete intestinal obstruction. Whether we ought to delay or immediately take active measures, will depend upon the site and type of obstruction, and particularly upon the acuteness of the symptoms. Even should spontaneous cure occur, it would only be a respite, hence there is no reason for long delay. It is true, however, that the results of operations for tubercular and carcinomatous strictures are only temporary, and in cases of multiple strictures a fatal termination is unavoidable.

Intestinal obstruction by fæcal impaction is not amenable to surgical treatment. Here the internal measures already described (purgatives, rectal injections of oil, soap and water irrigations, intestinal faradization or galvanization) are in place. Unfortunately, the diagnosis is often so extremely difficult that an operation may be necessary. After the diagnosis has been established, even in the severe forms, internal treatment alone is indicated.

Unless the intestinal paralysis is reflex in character (apparent reduction of external hernia, inflamed, undescended testicle, etc.), and its cause can be removed by operative or other methods, *intestinal paralyses* are to be treated as expectantly as possible. A serious result may often be averted by intestinal irrigation, electricity and purgatives. If these do not succeed, the best procedure is the formation of an artificial anus as high up as possible.

In the foregoing we have described the indications for operative treatment of intestinal obstruction. Discussion of the operative technic belongs rather to surgical literature.* We shall briefly call attention to a few of the principal points involved.

In every case of obstruction the aim of surgery is undoubtedly to remove the cause, and to attempt a permanent cure by laparotomy. Unfortunately there are often exceptions to this ideal result. Either the strength of the patient is insufficient, or the surroundings render radical operation impracticable, or existing conditions are so complicated that direct removal of the hindrance is too dangerous. In these cases enterostomy is the operation adopted by almost all surgeons.

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* An excellent *résumé* by E. Graser may be found in Penzoldt u. Stintzing's Handbuch, Bd. iv.

In the Congress for Internal Medicine, 1889, Schede[95] warmly advocated enterostomy. From his experience with two cases, he pointed out that the formation of an artificial anus might not only be a palliative but a curative operation. On the other hand, von Oettingen[96], from more extensive statistics, showed that enterostomy has not been successful in any case of volvulus with axial torsion, or in severe incarceration and strangulation; but that it had succeeded in intestinal kinking. In acute invaginations also, Rydygier[94], Gibson[93], and Ludloff[92] would limit the formation of an artificial anus to those patients whose strength does not allow of resection of the invagination. If the condition of the patient improves, a radical operation is later indicated.

From the above description it can be seen that there is a distinct gap between the therapeutic principles of the physician and of the surgeon, which will only be filled when the number of surgical cures becomes much greater than the medical. Excepting with a few surgeons, this at present is not the case. The changes produced in the obstructed intestinal segment and the resulting serious general condition develop so rapidly, that, to employ a common expression, the surgeon, even though operating at the earliest moment, generally operates too late.

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CHAPTER XIX

TYPHLITIS, PERITYPHLITIS (APPENDICITIS)

Preliminary Remarks.—Typhlitis is an inflammation of the cæcum and the surrounding peritoneum. Perityphlitis or appendicitis[*] is an acute or chronic inflammatory process, which originates in the vermiform appendix, and may remain strictly localized or spread to the surrounding parts.

At the present day we believe that in these inflammatory affections the cæcum is much less involved than the appendix. Thus the long-forgotten teachings of Louyer, Viller, Nay (1824), Mèlier (1827), Grisolle, and others,[†] which were so obstinately and successfully opposed, are now vindicated. From Talamon we gather that Mèlier had evidently foreseen the possibility of removing a diseased appendix. He plainly stated: "If it were possible to diagnose these affections with certainty we might conceive of the possibility of curing them by means of operation. Perhaps some day this result may be achieved."

Only very recently have the old mistaken views concerning appendicitis been overthrown. To a very great extent this advance is due to modern surgery. As in many branches of gastro-intestinal diseases, here also the autopsy *in vivo* and *post-mortem* have borne rich fruit (Ribbert, Zuckerkandl, Matterstock, H. Einhorn, and others). Internal medicine slowly yielded to modern views, and has since added considerably toward extending this new field. The excellent article of Sahli[1] and the impressive discussion pertaining thereto have accomplished much toward this end.

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[*] We will employ the terms perityphlitis and appendicitis, though the latter is etymologically not exactly correct. Because of its peculiar pronunciation, skolikoiditis (from σκώληξ, worm), the name introduced by Nothnagel, will probably not come into general favour. The term epityphlitis, recently suggested by Küster, though sounding better than skolikoiditis, is also objectionable, since the appendix is not always upon, but may be behind, below, above, or to the side of the cæcum.

[†] Compare the interesting historical development of this question by Talamon, Appendicite et Perityphlite, Paris, 1892; and by Grobe, Pathologie und Therapie der Perityphlitiden, Greifswald, 1896.

Surgery soon dominated this new field. In the middle of the eighties, with the progress and development of aseptic methods, the operative treatment of appendicitis made rapid advance in England and America, and soon obtained brilliant results.

Gradually our ideas of this affection became moderated, and the early radicalism was somewhat modified. Indications for internal and for operative treatment began to be compared, the results of internal and surgical treatment given their proper value, and the occasional bad effects of the operation (fistulæ, abdominal sinuses, herniæ, adhesions, etc.) were considered. Both scientifically and practically appendicitis was made the boundary between medicine and surgery. Before proceeding to a consideration of the diagnosis and treatment of this affection we shall, in what follows, state as briefly as possible its present status, in so far as it concerns the clinical view of the disease.

Typhlitis.—Does it really exist? Has stercoral typhlitis the significance attributed to it since Alber's time? Views differ; a few authors (among the modern writers I cite only Talamon*) go so far as to deny the occurrence of typhlitis. We may discuss these conditions in various ways—e. g., by referring to pathological anatomy, to clinical observations, or to the results of the many surgical operations.

Pathological anatomy has long dealt with inflammatory processes of the cæcum. Ulcerative conditions, particularly, have been known since autopsies have been systematically performed. Usually, however, these ulcerative conditions were quite different from those now in question. They were either typhoid, dysenteric, tubercular, stercoraceous, or actinomycotic. As already pointed out in the more extensive discussion of these ulcerations, they are by no means limited to the cæcum, but occur as often in the rectum, the flexures of the colon, and the sigmoid flexure. Indeed, it is surprising that, excepting the rare cases of pericolitis and sigmoiditis, inflammatory processes similar to typhlitis are not found more often in other segments of the large intestine. Etiologically, besides ulcerations, we must also consider foreign bodies (needles, etc.) and neoplasms (particularly cancerous and tubercular).

What is the status of clinical teaching regarding typhlitis? The symptoms of typhlitis are described as follows: Obstinate constipation, tympanites, pain and sensitiveness in the right iliac fossa, development of a sausage-shaped fæcal tumour corresponding to the position of the cæcum (*boudin stercoral*), moderate fever, and increased indicanuria. A perforation of the cæcum may occur, and death from peritonitis follow.

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* Loc. cit.

It must be admitted that in itself this picture is characteristic, but modern research has shown that it cannot be distinguished from disease processes which originate in the appendix. There is not *one* symptom which might not also be present in an appendiceal inflammation. *We cannot present an absolutely individualizing picture of typhlitis, and at the present time it is impossible to symptomatologically separate appendicitis and typhlitis.* The results of operations, however, or, as Nothnagel once appropriately called it, the results of "biopsy," can only determine the diagnosis during life.

Increased observations have demonstrated that, though rarely, isolated inflammatory processes may affect the cæcum or its vicinity without involving the appendix. Such observations have been reported by Harley[2], Mariage[3], Curschmann[4], Lennander[5], Porter[6], Krönlein[7], Menley[8], Meusser[9], and others. I agree with Borchardt[10] that some of these observations will not stand critical investigation, but among these there are several (I will only mention the case of Lennander, where no appendix at all was to be found) which need no forced interpretation, and which prove the occurrence of typhlitis.*

The etiology of typhlitis shows certain variations. There may be fæcal impaction with suppuration, or there may be adhesions; sometimes there are ulcerations of the most varied kinds, with more or less evident perforations. These variations prove that typhlitis by no means gives the simple clinical picture that appearances would warrant. It is necessary to give up the idea of a purely stercoraceous typhlitis, for impacted fæces per se rarely produce typhlitis; foreign bodies, ulcerations, adhesions, and fixation of the cæcum may also cause inflammation of the cæcum.

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* Grohé (loc. cit.) also reports an observation which belongs here. In a paralytic who died of broncho-pneumonia, the cæcum was filled with fæcal masses. In the portion of the intestinal wall opposite the mesenteric attachment there was found a slight ulceration of the mucous membrane of the size of a half dollar, which could only be explained by fæcal impaction.

Perityphlitis (Appendicitis).—It is now an absolutely established fact that the inflammatory processes which start in the appendix greatly preponderate, and that *the great majority of diseases until recently designated as typhlitis, perityphlitis, and paratyphlitis likewise originate in the vermiform appendix.* This fact is supported not only by anatomical observations (Matterstock, Fenwick, H. Einhorn), but also by the great number of observations of the foremost surgeons (Roux, Sonnenburg, Kümmell, Lennander, Körte, Schede, Rotter, McMurtry, Reginald Fitz, McBurney, Fowler, Treves, Beck, Kelynack, Dunn, and others). The absolute agreement of these surgeons relieves us of the necessity of presenting isolated statistics. The question arises as to which are the most frequent causative factors.

To answer this, a brief anatomical description of the relation and position of the appendix is necessary.

The length of the appendix varies from 2½ to 24 centimetres, the average being about 9 centimetres. Its thickness is about that of a goose quill. In the newly born the proportion of the length of the appendix to the large intestine is as 1 to 10; in the adult, 1 to 20. Like the cæcum, the appendix has a mesentery (mesenteriolum, mesovermium) in which the nourishing blood and lymph vessels course. The cæcal artery, the lowest offshoot of the superior mesenteric, gives off a branch to the mesovermium—the appendicular artery; the latter runs along the mesentery parallel to and a few millimetres away from the appendix, giving off branches to that organ. The appendicular artery is a terminal artery (in Cohnheim's sense), and this accounts for the severe changes which may develop in the course of apparently mild cases of appendicitis. The mucous membrane of the appendix contains cylindrical cells, Lieberkühn's glands, blood and lymph vessels, and is distinguished from the other intestinal segments by its striking abundance of lymph follicles. According to Ribbert[11], the latter vary very much with the age. In childhood the lymph follicles are very large and lie closely together; after the twentieth year they decrease in size and become more separated.

At the insertion of the appendix the so-called valve of Gerlach, or, according to Grohé, more properly the valve of Merling, is sometimes present, though frequently absent (Clado, Lafforgue). This structure, however, cannot prevent fæces from passing into the appendix.

Being a functionally unnecessary organ, the appendix presents involution changes with increasing age (Ribbert). After disappearance of the epithelium there develops a slow proliferation of the connective tissue of the mucosa, while the submucosa and muscularis retain their structure. By this process the lumen of the appendix becomes gradually narrowed, and in one third of all cases absolutely obliterated.

The relation of the peritoneum is important to an understanding of the anatomical changes. As modern observation (Bardeleben, Luschka, Tuffier) has shown, the entire circumference of the cæcum is covered by peritoneum, so that in more than 96 per cent of all cases the appendix lies intraperitoneally (Maurin[12], Bryant[13], F. Von Sydow[14]). The appendix may accompany the cæcum in its various changes in position. We will discuss this point more fully in the diagnostic section.

The manner of attachment of the appendix to the cæcum is of great surgical interest, and, as Krausshold[15] has long since shown, may vary considerably. The most frequent insertions of the appendix are internally to, behind, below and in front of the cæcum, and finally in the small pelvis (Bryant).

The causes of appendicitis are direct and predisposing.

Of the *direct causes* fæcal concretions play an important part. According to Ribbert[11] they occur in 10 per cent of appendicitis; according to Renvers[16], Treves[17], and Murphy[18] in about one third of all cases; according to some authors (Matterstock) they produce 50 per cent of the cases.

Other foreign bodies are found, but they are more rare than coproliths, being present in about 2 per cent of all cases.

The presence of fæcal concretions (which, by the by, have only a central nucleus of fæces, the remainder consisting of several layers of mucus (Ribbert[11])), by no means explains the entire etiology of appendicitis, for there are many cases in which these concretions are not present, but in which the appendix contains mucus or fluid fæcal masses. In view of this, we must ask whether fæcal concretions really play a considerable part in the etiology, or only favour the development of an inflammation. This question has been variously answered. In my opinion the concretion acts only as a predisposing factor in conjunction with other conditions soon to be considered.

We have already described the peculiar structure of the appendix, its abundance of lymph follicles, the absence of smooth muscle fibres, its extraordinary length in proportion to its narrow lumen, its tendency to changes of position and form, and, finally, the absence of anastomoses in the appendicular artery. All these circumstances favor catarrhal inflammation, which, no matter what its cause, leads to *increase* and *stagnation of secretion*.

The chief cause of appendicitis without doubt lies in the stagnation of its secretion. The pathology of other organs demonstrates the harmful effect of stagnating secretions in hollow viscera. In the gastro-intestinal canal they are the main cause of severe nutritive disturbances; in the gall bladder they are to a great extent responsible for formation of stones; and in the course of a surgical wound the absence of sufficient flow of secretion constitutes one of the most important complications. Evidently all this also applies to the appendix. As long as there is free and regular communication between the mucous membrane of the appendix and cæcum, even though virulent bacteria be present, no disturbance will follow. The moment, however, the communication ceases, and the outflow of secretion is prevented either by fæcal concretions, catarrhal swelling, partial obliteration, or by compression or adhesion of neighbouring organs, decomposition of the contents, inflammation, ulceration, gangrene, and suppuration occur.

As demonstrated by the careful studies of Tavel and Lanz[19], Eckehorn[20], Morris[21], and others, bacteria, especially the bacterium coli communis, undoubtedly play a prominent part in the above processes. The destructive powers of the bacteria are only exercised when a favourable medium for considerable development, and perhaps also for their transplantation, is offered.

As already mentioned, this medium is furnished by the stagnation of secretion, by loss of epithelium, by erosions, or by catarrhal or pressure ulcerations, which, owing to the poor blood supply of the appendix, develop only too easily.

If, therefore, the fæcal concretions are given their proper, rather large predisposing part in the etiology of perityphlitis, and if we do not underestimate the influence of the bacteria natural to the appendix, the direct development of the inflammatory process must be sought for in the peculiar anatomical form and structure of the appendix, which produce and make possible the deleterious action of both the above factors.

Another circumstance which speaks in favour of the above view is the infrequency of perityphlitis in childhood. In the statistics of Matterstock, of 474 cases of perityphlitis, there are only 46 cases between the ages of one and ten years. Out of 228 cases Fitz gives 22 of the latter age, and out of 130 Sonnenburg only 26. Still more convincing are the statistics of the pediatrists. From the excellent monograph of Karewski[22] on perityphlitis in childhood, we find that Henoch, during the years 1890–1894, among 3,486 sick children, saw only 2 cases of appendicitis. Baginsky, in 1890–1891, of 494 cases, saw none; of 415 cases in 1891–1892 he also observed none; of 1,692 cases in 1892–1893, he observed but 3; in 1893–1894, of 2,234 cases, only 4; in 1895, of 2,580, 8 perityphlitis cases; altogether 15 cases out of a total of 7,413 diseased children.

Again, from H. Einhorn's statistics from the Pathological Institute of Munich, the number of cases of perityphlitis in proportion to total autopsies was 5 per 1,000, the cases in children being 2 per 1,000. Since the diagnosis of this affection in children is quite difficult, this proportion is rather too large than too small. In this connection Nothnagel's[23] statistics are also of great value. Of 44,940 autopsies performed in the Vienna General Hospital in the years 1870 to 1896, there were 148 cases of peri- and paratyphlitis (0.3 per cent). Of these 148 cases only 2 were in children between one and nine years of age.

The explanation of these facts is furnished by the investigations of Steiner[24], Ribbert[11], and Zuckerkandl[25]. These observers found partial or total obliteration of the appendix with increasing age, while obliteration was extremely rare in children, particularly before the fifth year. In my opinion the large size of the lumen of the appendix, whereby accumulations of secretions and pressure necrosis are prevented, is of great importance in explaining the rarity of appendicitis in children.

Besides these essential factors certain predisposing circumstances play an undoubted part. Talamon[26] lays stress on heredity. Some families present a predisposition to appendicitis. Sahli also mentions such cases. Traumatism is mentioned as a factor by some authors (Coley[27], Small[28], Körte-Borchardt[10], and others). Various observers give chronic constipation as a predisposing cause. In 209 appendicitis cases Fitz[17] found constipation only 38 times.

I believe we must differentiate between acute and chronic recurring appendicitis. According to my experience chronic constipation is quite often a factor in the latter, but much less so in the acute form. Treves[17] regards bad teeth and insufficient chewing of food as frequent causes of appendicitis. Dyspeptic conditions are said to favour this disease. In English literature particularly, rheumatism and gout are mentioned as predisposing to an attack.

Recently Golubeff[29] has directed attention to the *frequent simultaneous* occurrence of perityphlitis in Moscow, and has therefore attributed to it an epidemic character. From a study of cases occurring in Erlangen during a period of nine years, Penzoldt[30] could not substantiate this conclusion. At times I have seen numerous simultaneous perityphlitis cases in Berlin; a number of factors may here have acted together.

We might further increase the list of accidental causes of appendicitis, and add that affections of the female genitals also predispose to appendicitis. All these facts can not overthrow the real practical etiological considerations that we have above attempted to establish.

Perityphlitis of *tuberculous* or *actinomycotic* origin presents a certain individuality. The former not infrequently occurs as a latent symptom of general and particularly of intestinal tuberculosis. On the other hand, primary tuberculous disease of the appendix with a characteristic picture of appendicitis is exceptional. From a very large material Sonnenburg reports but 3 cases of this kind. Borchardt[10] also reports 3 cases occurring in Körte's hospital service. Karewski[22] has operated on 4 cases in children, in 2 of which he thinks the disease was a primary isolated tubercular appendicitis. According to Borchardt, tuberculosis may produce perityphlitic abscesses which rupture externally and cause cæcal fistulæ.

Actinomycotic appendicitis is more infrequent. In his monograph Sonnenburg could collect only 12 cases of this kind. Karewski[22] reports a further case.

In conclusion, we wish to discuss the relative frequency of this disease in the various periods of life and its relation to sex.

We have already dwelt upon the relative infrequency of appendicitis in childhood. Numerous statistics (Matterstock, Fitz, Nothnagel, Sonnenburg) demonstrate that the second and third decades of life present a very striking predisposition to appendicitis. According to the extensive tables of Matterstock, 63 per cent of all cases of appendicitis occur during these periods. Except those of H. Einhorn, the remaining statistics indicate a similar conclusion.

Regarding the relative frequency in the two sexes, according to the tabulations of Volz, Bamberger, Matterstock, Sonnenburg, Rotter, Nothnagel, Fitz, Pravaz, and Fenwick, appendicitis is much more frequent in men than in women, while others (Einhorn, Lennander, and Kümmell) could discover no material differences in frequency. Nothnagel correctly points to the greater frequency of appendicitis in the male even in childhood. This is also evidenced by Matterstock's extensive tables (51 male children to 21 female).

Symptomatology and Diagnosis

A. Acute and Chronic Typhlitis

We have already briefly described (page 432) the symptoms of acute typhlitis, and have mentioned pain and sensitiveness to pressure in the cæcal region, tympanites, palpable fæcal accumulation, and fever. This description presupposes a simple stercoraceous typhlitis. We have as yet no clear clinical picture of the remaining forms of typhlitis, at least not of the acute forms. This is evident from the fact that in almost all operations in which typhlitis was found the diagnosis of appendicitis had been made. It is therefore idle to lay down diagnostic rules and principles for these other forms of typhlitis. But in the present state of our knowledge even the differentiation between stercoraceous typhlitis and appendicitis must be made with the greatest reserve. Under the following circumstances the diagnosis of stercoraceous typhlitis might be ventured. Sudden, obstinate constipation, moderate sensitiveness over the cæcum, mild fever or none at all, and absence of severe general symptoms. The intensity of the spontaneous pains appears to me irrelevant. Objectively, we should be able to palpate a fæcal tumour characterized by its compressibility and perhaps extending high up along the ascending colon. There is a dull percussion note over the tumour. Clinical course: Immediate disappearance of the tumour and of all symptoms after a laxative or enema. Only in the presence of a clinical ensemble as well marked as this can the diagnosis of [stercoraceous] typhlitis be made with a fair degree of probability. So typical a picture is certainly not frequently seen in practice, because the majority of patients have already taken laxatives before medical advice is sought. Even in such typical instances it is impossible to positively differentiate the affection in question from appendicitis or appendicular colic. We shall recur to this point in the section on differential diagnosis.

Under appropriate circumstances *chronic typhlitis* can be more easily diagnosticated than the acute form. The absence of violent initial symptoms, the very slow onset, the palpability of a resistance in the cæcal region, all point toward the diagnosis. Since chronic typhlitis is usually tuberculous, dysenteric, or carcinomatous in character, the previous history or other clinical phenomena may give important diagnostic data. The presence of stenotic symptoms will often serve to make the etiology positive. As proved also by surgical experience, it is very difficult to diagnosticate adhesions about the cæcum (*perityphlitis sensu strictiori*). Even admitting the very rare instances in which the adhesions can plainly be felt, it will be scarcely possible to prove their origin from the cæcum. At most this can only be surmised.

B. Perityphlitis (Appendicitis)

1. *Acute Perityphlitis*

In many instances the diagnosis of acute perityphlitis is as easy as that of croupous pneumonia or of an acute monarthritis. Conversely, however, the clinical picture may be so complicated and atypical that, despite great experience and ability, error is unavoidable. The statistics of Nothnagel, already given (page 436), derived from the very extensive material of the Vienna General Hospital, contain a number of such diagnostic errors. These cases are the more instructive because they have been under most careful clinical observation.

The diagnostic difficulty lies in another direction. Operations have taught us the numerous and various changes which the appendix may undergo: the diseased process may be limited to the mucous membrane, or to the peritoneal coat, or may affect both; rupture may take place into a preformed encapsulated space, into the general peritoneal cavity, præperitoneally, retroperitoneally, into the intestines or other hollow viscera. Even during laparotomy it may be impossible to distinguish these various forms of appendicitis. It is absolutely necessary, however, to understand the principal types, and to be able to recognise the most important clinical complications of the disease.

The variations in the clinical picture of appendicitis have led to its classification. Sonnenburg[31] was the first to establish and methodically carry out such clinical divisions. His classification is as follows:

1. Simple, catarrhal appendicitis, with its acute, chronic, obstructive, and cystic forms.

2. Perforative appendicitis: (*a*) with periappendicitis, (*b*) with general peritonitis.

3. Gangrenous appendicitis: (*a*) septic peritonitis without perforation, (*b*) circumscribed or diffuse peritonitis accompanying beginning perforation.

Rotter[32] has divided perityphlitis more simply into the circumscribed and the diffuse forms. Kümmell[33] divides the cases into the mild, moderate, and severe—an arrangement which is followed by Körte.

Naturally all these classifications are schematic, and give only an incomplete idea of the various phases and clinical courses of appendicitis. Rotter's arrangement seems to me the simplest and least prejudicial, but it also incompletely represents the varieties of the disease. With much hesitation, we shall follow Sonnenburg's nomenclature, discarding only those subdivisions which either go too far or not far enough.

Though such differentiation cannot be made with certainty, we shall also distinguish between simple perityphlitis and appendicular colic.

(*a*) SIMPLE PERITYPHLITIS

The most prominent symptoms are sudden onset; acute pain in the ileo-cæcal region; sensitiveness to pressure in the region of the appendix, and eventually of the tumour; gastric disturbances; the condition of the pulse and the temperature.

Each of these symptoms requires detailed consideration.

1. *Sudden Onset.*—The patients are usually attacked in the midst of general good health. Occasionally, constipation or diarrhœa precedes the attack. The attack very soon becomes so marked that the patients are forced to take to bed.

2. *Pain.*—This is the most characteristic symptom, and rapidly reaches its greatest intensity. Adults can usually localize the pain quite well, but children complain of general stomach ache. Some patients locate the greatest area of pain in the centre of the abdomen, in the umbilical or epigastric region.* The pain is continuous, and shows slight or no remissions. It may radiate to the right thigh, the back, the testicle, or the bladder. In the last instance bladder symptoms may be present, and there may even be retention of urine. The respiration is a useful gauge for the intensity of the pain. In well-marked cases the breathing is rapid, superficial, and costal; the patients anxiously avoid deep inspiration. The patient feels most comfortable when lying quietly on his back; every movement increases his pain, absolute rest decreases it.

3. *Sensitiveness.*—Sensitiveness to pressure over McBurney's point is the most valuable and reliable of the objective symptoms. The sensitiveness varies in intensity, but in the beginning is fairly

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* [Hartley[77] states that only in one fourth of the cases is the initial pain referred to the right iliac fossa.—TR.]

well localized. As Fowler[34] points out, there is a certain amount of rigidity on palpation of the right rectus muscle. He also calls attention to an interference with the function of the abdominal respiratory muscles, evidently caused by the above-mentioned superficial and rapid breathing. Regarding the possibility of palpating the inflamed appendix, to which Edebohls[35] in particular refers, views differ. Sonnenburg, whose opinion is certainly of great value, says "that the thickened appendix cannot be palpated during acute attacks, but may be palpable in the intervals." Fowler[34] speaks of "exceptional identification in cases of chronic appendicitis."

Admitting the possibility of feeling the appendix, this fact is on the whole of small practical significance. A circumscribed painful area in the ileo-cæcal region is a sufficient clinical symptom.

4. *Perityphlitic Tumour.*—Under appropriate treatment the sensitiveness may disappear in a short time, or may increase with the onset of high fever and the development of a perityphlitic tumour. Of what does this tumour consist? According to Sahli[1], it is mainly composed of a thickening of the intestinal wall, the visceral and parietal layers of the peritoneum, and the fascia transversalis of the abdominal muscles; in addition, there are fibrous adhesions of the intestines, not infrequently thickening of the omentum, and stagnation of the intestinal contents.

In general, even small tumours of the iliac fossa are readily palpable. In perityphlitis, because of the rapid onset of intestinal paralysis, the intestines become distended with gases and the tumour is thereby brought nearer to the surface. The tumour is felt either as a circumscribed, easily defined mass, or as a diffuse, doughy swelling (Roux). In other cases, especially when there is marked meteorism, or when the appendix is situated behind the cæcum, recognition of the tumour by palpation is difficult. In all these cases rectal or vaginal examination is of great value, especially since the appendix is sometimes situated low down in the small pelvis and may there give rise to abscesses.

In doubtful cases exploratory puncture may render important diagnostic aid. At present the views regarding the value of this procedure are contradictory. Some prominent surgeons (Sonnenburg, Roux, Karewski, Fowler, and Treves) have entirely given up puncture, while Körte, Borchardt, Lauenstein, as well as the majority of internal practitioners (v. Leyden, A. Fraenkel, Fürbringer, Renvers, Curschmann, Sahli, Nothnagel, Penzoldt, and others), strongly recommend it.* Summing up the experience of the latter, we may say that exploratory puncture is usually not a dangerous procedure. Puncture of the intestines cannot always be avoided. Penzoldt† believes he has occasionally punctured the bowel without causing any trouble. Karewski saw two injuries of the intestines from puncture during operation, which, he states, were not followed by deleterious effects. In another case Karewski ascribes a peritonitis (and subsequent operation) to puncture, but I am not convinced of the correctness of his statements.

In my opinion, we should puncture only when an abscess is suspected and its presence cannot otherwise be determined. I do not consider it advisable to employ exploratory puncture for the purpose of showing the patient the necessity of an operation, for the patient may recover without operation—a fact which would place the physician in a rather awkward predicament.

Regarding exploratory puncture, Penzoldt[30] has laid down several excellent rules, which we here reproduce.

> The needle must have as large a lumen as possible and still be fine, long, and strong enough to withstand bending. Instead of the point having the usual lancet-shaped tip and being sharpened laterally, it should be round and only sharpened at its extreme tip, so that the lower end of the needle is not larger than the upper, thus avoiding an unnecessarily large puncture.
>
> If carbolic acid, or ether and alcohol, have been used to disinfect the needle, sterilized water should be drawn through the needle directly before puncture, because any portion of the above disinfectants remaining in the syringe may precipitate the albumin of the fluid aspirated and thereby give rise to difficulties. (For the better conservation of the cell elements, I draw sterilized salt solution through the syringe directly before and after aspirating.) The needle must fit the barrel of the syringe well. The puncture is made at the point of greatest resistance and dulness; if necessary, several punctures may be made. After puncture of the abdominal muscles, the piston is slightly withdrawn; if nothing be aspirated, the needle is pushed in somewhat deeper, the piston being retained in the same position. The piston is then drawn out somewhat further, and if again nothing be aspirated, the needle is thrust deeper, the piston then further withdrawn, and so on till the piston has been drawn out its entire length. The syringe should be large enough to hold 2 to 3 cubic centimetres, so that a large area may be explored through one puncture. The needle is steadied slightly with the left hand, so that it may follow the movements of the abdominal muscles. Puncture must never be performed without a microscope near at hand, so that clear fluid may be imme-

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* [Exploratory puncture is a procedure not generally practised in the United States; it is rare to find it mentioned in text-books and monographs, and when mentioned it is usually condemned.—Tr.]

† Loc. cit., p. 671.

diately examined for cellular elements. The small amount of fluid hidden in the needle may be easily identified. (Where, as in private practice, especially in the country, a microscope is not handy, it is better to send the entire syringe in a properly stoppered container for examination.)

Compared with the above diagnostic data, *percussion* is of secondary importance. It is clear that in the presence of a distinct exudate the percussion is dull or dull tympanitic; but if the exudate be situated very deeply, percussion will scarcely be of diagnostic aid.

The same is also true of *auscultation*. For the purpose of differentiation between simple and septic paralysis of the intestinal muscles, Richardson[36] states that intestinal sounds are present in the former and absent in the latter. The "rumbling" sound described by Naumann[37] as characteristic of abscess does not appear to have been observed by others. In a case without suppuration Borchardt* discovered this sound, which he considers an ordinary intestinal noise.

5. *Gastric Disturbances.*—These consist in loss of appetite and occasionally of vomiting, especially in the beginning. In children vomiting is often the ushering-in symptom, and may be productive of diagnostic errors—e. g., acute dyspepsia, intestinal catarrh, etc. In the further course of the disease the vomiting generally ceases, but should it persist and become more marked, suspicion of severe invasion of the peritoneum must immediately be aroused. Should the vomiting become fæcal in character, we must think of the possibility of mechanical or paralytic intestinal obstruction accompanied by peritonitis.

6. *Temperature.*—Temperature is of great diagnostic and prognostic importance. In simple catarrhal appendicitis the temperature is low and falls rapidly, but in the purulent and perforative forms it very soon becomes quite high, and remains so for a longer or shorter time. To avoid repetition, we shall now discuss the course of the temperature in all the different varieties and stages of appendicitis. Rotter[33] has made a careful study of the temperature in the various forms of appendicitis, and although, as must be expected from its protean type, the disease contradicts all rules and experience, the fever curves of Rotter are very important for the recognition of the status of individual cases. Rotter arranges the cases, according to their temperature, into five groups:

The first group is ushered in by marked fever (up to 40° C. and over), with or without a chill; after 3 or 4 days there is a tendency to defervescence. These cases are characterized by a smooth and rapid convalescence.

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* Loc. cit., p. 330.

In the beginning cases of the *second group* are not distinguishable from those of the first. The fever, however, lasts longer; after the fifth day the temperature is not higher than 39° C. These cases also recover, though sometimes only after operation.

The third group is characterized by the fact that after the fifth day the temperature remains above 39° C. From the time of onset these cases present more or less high fever, and also slight remissions followed by increase of temperature. Most of these cases generally run a severe course and sooner or later require operation; of those not operated upon some recover and others die.

The fourth group includes those which present a remittent type of fever. The initial fever is followed by defervescence, but after a few days the temperature again rises. This second rise indicates suppuration. Most of these cases required operation. Of those not operated upon, one seemingly recovered, another died of diffuse peritonitis.

In the fifth group, which includes those with diffuse peritonitis, temperature has no special significance. It may be high, normal, or even subnormal. If a circumscribed abscess ruptures into the general abdominal cavity, there is a sudden fall of temperature, often subnormal and accompanied by collapse.

7. *Pulse.*—Next to temperature, the frequency of the pulse and its possible irregularity are of the greatest value. There is a special significance in a disproportion between temperature and pulse. From numerous operations we know that severe peritonitis may exist without any rise of temperature. It is in these cases, of which I have observed several, that the character of the pulse becomes the only diagnostic and prognostic indication.* On the other hand, Lennander,† Karewski[22], and others, have shown that in the severest cases of progressive, suppurative, fibrinous peritonitis, both temperature and pulse may be almost normal (Mikulicz). A distinct and continuously irregular, and at the same time very small pulse, is almost always an ominous sign.

Finally, we would mention an interesting phenomenon pointed out by Mannaberg[38], viz., the accentuation of the second pulmonary sound. He discovered this symptom in cases of perityphlitis oftener than could be accounted for by mere accident.

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* [In the United States the pulse is generally regarded as the most valuable single prognostic and therapeutic indicator in appendicitis.—Tr.]

† Loc. cit., p. 27.

(*b*) Appendicular Colic

Under the name of *colique appendiculaire* Talamon[*] first described a clinical picture characterized by the following symptoms: The patient is suddenly attacked by very severe pains, occasionally accompanied by vomiting. The region of the appendix is extremely sensitive to pressure; fever is either very moderate or entirely absent; a tumour never forms. The attack passes off very rapidly. Spontaneously, or after morphin, the patients are absolutely well at the end of twenty-four to thirty-six hours. Talamon explains this condition by supposing that a fæcal concretion is wedged into the appendix and thereby causes violent muscular contractions; finally the concretion falls back into the cæcum, similar to a gall-stone falling back into the gall bladder after having reached the cystic duct.

Von Hochstätter[39], Caspersohn[40], Goldbach[41], Treves[17], Nothnagel, Sonnenburg, and A. Pick[42] have described such cases. I have seen a large number of these cases, but in view of the simplicity of the clinical picture I shall not describe any. In my experience the patients are generally individuals who suffer frequently from such attacks (every four to six weeks or oftener). In the interval they are absolutely well, but the attacks may finally become continuous, and assume the character of chronic perityphlitis.

All authorities agree upon the symptoms, but there is a diversity of opinion regarding the origin of this disease. Treves[17] denies the possibility of muscular contraction of a healthy, let alone a diseased, appendix, and characterizes this theory as "wholly ridiculous." Nothnagel admits the possibility of spasmodic contraction, but does not consider it proved that coproliths wedge themselves into the appendix. Monod and Vanvers[43] express a somewhat similar opinion. Opposed to these theories is the cited case of Goldbach, observed in Wölfler's clinic in Prague.

The patient was a sixteen-year-old scholar, who for one year had suffered from jaundice and severe colicky pains under the right free border of the ribs. He never had fever or vomiting during the attack. Always had obstinate constipation. Later, pains were present in the evening and absent in the morning He now has pain in the ileo-cæcal region, localized directly over McBurney's point. No concretion was ever found in the stools. Palpation shows an oval, fairly soft tumour (cæcum ?), over which may plainly be felt a second longitudinal sausage-shaped tumour. The entire mass appears movable, and is felt either in the right hypochondrium or in the lower abdominal region. Liver is not enlarged. At the operation two small fæcal stones were found in the cæcum. When these were pressed toward the appendix they easily slipped in, and could just as easily be forced back into the cæcum. The appendix was absolutely normal. Extirpation of the appendix. Cure.

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[*] Talamon, Appendicite et Perityphlite, Paris, 1892, p. 25 et 111; Colique appendiculaire médecine moderne, 1890, p. 837.

This case at least proves the possibility of foreign bodies slipping into the appendix and again falling back into the cæcum. It is very questionable whether this is a constant or only frequent occurrence. From the fact that in most autopsies [and operations] for recurring appendicitis (which the above case greatly resembles) slight changes are found in the appendix, it would seem that these are after all very mild forms of simple catarrhal appendicitis.

(*c*) PERFORATIVE PERITYPHLITIS

Pathological anatomy and the results of operation agree that perforations occur very frequently in appendicitis. It is therefore important to be able to diagnosticate this complication. Sonnenburg gives certain symptoms as characteristic: Violent onset with high fever; severe abdominal pain beginning suddenly or immediately after a meal, and very soon localized on the right side; vomiting, accompanied by diarrhœa or constipation; small, frequent pulse; fever, rising rapidly and often ushered in by a chill; marked tympanites. Patient feels extremely ill. There is slight cyanosis and perspiration. Distinct resistance in the vicinity of the suppurative area.

The diagnosis of perforation, however, cannot be positively made from any of these symptoms, for suppurative appendicitis with tumour, severe general disturbance, high temperature, and rapid pulse may give a very similar picture.*

It will therefore be wiser not to attempt to make the diagnosis of perforative peritonitis, but to content ourselves with the diagnosis of suppurative appendicitis.

(*d*) DIFFUSE SUPPURATIVE PERITYPHLITIS

According to Rotter, diffuse suppurative perityphlitis originates in two ways: either as a purulent perityphlitis, in which the adhesions between the intestinal coils continue to extend, or by perforation of a previously encapsulated abscess, whose contents spread over the general abdominal cavity. In both cases the pus generally gravitates toward the lower right side of the pelvis and Douglas's

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* [A blood count might prove of great value in differentiating the two conditions. Marked leucocytosis would speak for suppurating appendicitis.—TR.]

cul-de-sac; if the process continue, the pus spreads to the left side of the pelvis, and from there over the different abdominal organs.

The diagnosis of the various phases of this variety may be extremely difficult. Temperature, pulse, and objective symptoms may be deceptive, and easily lead to false conclusions. As already remarked, if high temperature is present, it is irregular and remittent in character, becoming normal, again suddenly rising, etc. The most reliable symptom is the general appearance. The patients are usually markedly collapsed, depressed, apathetic, the features are pinched and sunken, and are pale, cyanotic, and without congestion; the eyes are staring; there is absolute sleeplessness and anorexia; the tongue is dry and cracked; occasionally there is singultus. In a word, the patient makes a septic impression. To this picture there are exceptions. The patient may feel well, sit up in bed, his pulse and temperature be properly proportioned and not high, and yet laparotomy will reveal a diffuse, septic peritonitis. In other cases, very grave symptoms are at first present; then suddenly the patient becomes better, "the suppuration becomes circumscribed" (Rotter). Contrary to expectation, the patient recovers, perhaps only after one or several abscesses have been opened. Numerous other varieties are seen in practice, but a schematic description is impracticable, because every case presents special peculiarities. We have here purposely given the clinical picture, but not the diagnostic criteria of diffuse perityphlitis, because after all the general impression created by the patient is the decisive diagnostic factor.

The diagnosis, and I may add the prognosis, often vary extremely, as the clinical picture changes from day to day. This should warn us never to give a prognosis too early in the disease.

2. *Chronic Perityphlitis*

Chronic or recurring perityphlitis is that form in which, after a longer or shorter interval, renewed attacks of the disease occur. The American surgeons (Bull[44], Fowler[34], and others) designate this form as "recurring" appendicitis, and distinguish it from those cases in which the acute attack is recovered from, but a sensitiveness to touch persists in the ileo-cæcal region. The last form has been called "relapsing" appendicitis, though some, like Fenger of Chicago, call it "postappendicitis."

The studies of surgeons, particularly Sonnenburg, Kümmell, Bull, Fowler, Körte, Treves, and Senn, have given us valuable information regarding chronic appendicitis. This disease may develop in many different ways. The most frequent mode is through the formation of partial obliterations (appendicitis obliterans, Senn) and strictures, with consequent stagnation of secretion, formation of cysts, and occasionally of empyema of the appendix. More or less extensive adhesions may develop about the appendix, and cause functional irregularities of the intestine, bladder, and female genitals, and produce pain and other disturbances. The mucous membrane of the appendix may be diseased, and small swellings and suppurations exist in or around the appendix; fæcal concretions are sometimes present. Under such conditions the appendix may perforate during a relapse.

If we limit ourselves to the diagnosis of a diseased process in or about the appendix, we will scarcely ever meet with any difficulty. The history may give us valuable information. More important, however, are the typical symptoms (pain, even while at rest, but increased by motion or straining; constipation) and the discovery of a circumscribed area of sensitiveness to pressure, and of infiltrations about the cæcum or appendix.

In recurrent perityphlitis the patients have no symptoms between the attacks. From time to time, either without recognisable cause or after strains, errors in diet, colds, or constipation, the pain recurs, a palpable tumour in the appendix region, accompanied by fever, nausea, and vomiting, appears, and in about one half the cases perforation occurs.

As Treves has pointed out, and as is generally known, the recurring attacks are usually not as severe as the primary one. In recurring appendicitis *perityphlitic abscesses* seem to develop very rarely. Talamon [*] records a case in which an abscess, necessitating surgical treatment, developed in each of four attacks.

In the vast majority of instances the proper diagnosis can be made from the symptoms above enumerated. The diagnosis is difficult only in those cases in which appendicitis must be distinguished from disease of the female genitals. We shall discuss this under differential diagnosis. On the other hand, it is almost impossible to diagnosticate the individual pathological conditions in and about the appendix; it would therefore be useless to enter into a discussion of the diagnosis of this point, which, moreover, is of little practical value.

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[*] Loc. cit., p. 151.

DIFFERENTIAL DIAGNOSIS

The diagnosis of a typical case of appendicitis is simple; it may become quite difficult when the disease runs an irregular course, or when the patient is first seen during the late stages of the disease. It is impossible to consider all the numerous differential possibilities; in the following we shall discuss only the important ones.

Simple appendicitis gives rise to the least difficulty. Mistakes may occur if the pain is atypically localized, or if the objective symptoms are entirely or almost entirely absent.

In the former instance (i. e., the atypical location of the pain) we must consider *biliary* and *renal colic*, and occasionally *mucous colic*. In most cases careful observation will scarcely leave room for doubt. According to Naunyn, the examination of the urine for indican may be of value. Naunyn[45] states that indican is never absent in appendicitis, while in biliary colic it is only occasionally present. As another important diagnostic fact, it must be mentioned that in cholelithiasis the posterior surface of the liver, between the tenth and twelfth dorsal vertebræ, two to three fingerbreadths to the right of the vertebral column, is sensitive to pressure. In nephrolithiasis, it will be necessary to carefully palpate the right kidney and to examine the urine.

In children, and sometimes in adults, appendicitis may be confounded with "febrile gastro-enteritis." Karewski[22], in particular, has directed attention to these errors, and every physician will appreciate his warning. If, as a result of this error, these cases are treated with calomel and castor oil, serious danger may arise. In children, in all cases of so-called febrile gastro-enteritis accompanied by acute pain, we should at once think of appendicitis. If we then err, there can never occur the severe consequences that may otherwise arise.

The diagnosis is far more difficult if the sensitiveness to pressure or an exudate is localized, not at McBurney's point, but at other places—e. g., in the right or left hypochondrium, the umbilical region, the left iliac fossa, etc.

The experiences of surgeons, particularly the oft-quoted topographical clinical studies of Curschmann, have shown us how the cæcum and the appendix may be found in various situations. In one case, close to the right costal border, Curschmann[46] found a hard superficial tumour the size of the palm of the hand. The tumour lay in front of the intestines, and was connected with the inner surface of the abdominal wall. Judging from its origin, position, and form, it was a peritonitic exudate. The patient died of general peritonitis. At the autopsy the cæcum was found turned up in front of the descending colon, with the appendix touching the liver (see Fig. 25).

I herewith present a similar case described by Rotter:

> A man, fifty-one years old, while under ambulatory treatment at Kissingen, became ill with gangrenous appendicitis. Eight days after the onset of the disease a tumour was found between the liver and the ascending colon. The most striking feature was the great mobility of the tumour; it could easily be moved across the middle line toward the left, whereas perityphlitic tumours in general are diffuse, and attached to the posterior abdominal wall.

The diagnosis rested between a tumour of the colon and of the kidney till an exploratory puncture showed feculent pus. The great mobility of the tumour was due to the fact that it was not adherent to the anterior or posterior abdominal wall, but rested upon the right side of the mesentery of the small intestine, the remainder of the abscess wall being formed by coils of adherent intestine. In displacing the tumour, the mesentery and adherent bowel moved with it.

In his Clinic of Cholelithiasis, Naunyn mentions similar instances. Almost all experienced surgeons (particularly Sonnenburg, Lennander, Fowler, Körte, and Riedel) have reported similar cases. The practical conclusion to be drawn is that, although an exudate or localized sensitiveness be found in other than in the typical situation, we must always keep in mind the possibility of appendicitis.

Cases of pericolitis and sigmoiditis (later to be described) show that inflammatory exudates, though indeed more rarely, may occasionally originate from other sources than the appendix.

Exploratory puncture may be of diagnostic value. The withdrawal of feculent pus indicates a perforating appendicitis.

It may be very difficult to differentiate between *inflammatory disease* of the *female adnexa* and appendicitis. The differentiation may be impossible when, as often happens, appendix and adnexa are simultaneously diseased. I have seen several cases of this kind. Accumulations of pus in Douglas's pouch may also give rise to diagnostic errors. Borchardt thinks that the differentiation can be made by examining the pus for bacterium coli communis, the presence of which would speak for appendicitis.

For further details of the subject the reader is referred to the monograph of Sonnenburg.

The differential diagnosis is quite difficult where the appendix is in the small pelvis and there gives rise to an abscess. In these instances the differential diagnosis between appendicitis and inflammatory disease of the adnexa can rarely be made.

Tumours, particularly of the cæcum, may be mistaken for chronic appendicitis, and, much more infrequently, for acute appendicitis. The tumours here to be considered are sarcoma, carcinoma, actinomycosis, tubercular tumours of the cæcum, occasionally also ileo-cæcal invagination.

For purposes of differentiation, Sonnenburg[47] recommends inflation of the intestines. In perityphlitic exudates the intestines become distended, in neoplasms they remain rigid. The mobility of a questionable tumour speaks decidedly against an exudate; but immobility does not speak against neoplasms, since they may be fixed by adhesions. Where symptoms of stenosis of the large bowel are present the diagnosis may be easy. If these be absent, the insidious course, the cachexia (in malignant tumours), the presence of blood or pus in the stools, will indicate the correct condition. Reports of cases show, however, that numerous errors are made which are only cleared up by operation or autopsy. (Compare with chapter on Intestinal Carcinoma.)

Finally, we have the rare occurrence of the inflamed appendix in a *hernial sac* (inguinal or femoral canal) which has resulted from fœtal maldevelopment. This may occur on the left as well as on the right side. These cases run their course as incarcerated herniæ, and are of great surgical interest. The diagnosis is almost impossible.

Appendicitis in the stage of *diffuse peritonitis* may cause considerable diagnostic difficulty. This originates under very different circumstances. In the first place, diagnostic confusion may be present when, in the midst of apparently normal health, or after obscure premonitory symptoms, signs of perforative peritonitis appear. It is difficult to determine the cause of the condition and the proper site for the surgical incision, upon which considerations the life of the patient may depend. In the chapter on Duodenal Ulcer (page 293) we have seen that not infrequently a perforation of the appendix is sought for, and autopsy reveals a perforating duodenal ulcer.

Regarding the differentiation between perforations of duodenal or gastric ulcers and perityphlitic abscesses, Marmaduke Sheild[48] ascribes great value to the fæcal smell of the pus and the gas bubbles formed; these would indicate disease of the appendix and cæcum. He also lays stress upon the reaction of the pus, which is neutral or alkaline in appendicitis, and acid in the others. Mistakes like the above will never be unavoidable, but they warn us not to operate till the site and nature of the perforation have been determined with some degree of certainty.

I believe that the prominence given to perforative appendicitis tends to keep all other etiological factors in the background.

What is true of duodenal ulcer is also true of gastric and the other numerous and genetically different forms of intestinal ulcers. The differentiation between perforating ulcer of the cæcum and of the appendix can only exceptionally be made. In sudden perforation this distinction naturally has no practical significance.

We are frequently called upon to differentiate between appendicitis and intestinal obstruction. In the following section we shall discuss chronic obstruction as a complication of appendicitis. In the cases there cited the diagnosis was easy, since the previous attack of appendicitis clearly indicated the original trouble. In acute cases the differentiation is much more difficult. (Compare also the chapter on Intestinal Obstruction, page 368.) The symptoms may be due to a variety of causes. The intestinal obstruction may result from reflex intestinal paralysis, from compression of a perityphlitic exudate, from kinking produced by adhesions, or from any other of the almost innumerable ordinary causes. If no palpable abscess be present, the history not definite, the temperature normal or almost so, the patient collapsed and vomiting fæces, I see no possibility of distinguishing between perityphlitis and obstruction. The situation is the more critical since the circumstances necessitate quick decision, and do not allow of careful and thorough examination and observation.

In these instances the laparotomy will clear up the diagnosis. We must again emphasize the value of a careful history, which may offer an etiological hint and enable us to reach the proper diagnosis. The history does not entirely guard against error, as proved by a case of Sonnenburg[47], in which the history indicated perityphlitis, but laparotomy showed obstruction by gallstones. It is hardly necessary to state that the above diagnostic difficulties do not always exist, and that when the symptoms are typical the diagnosis may be made at sight.

Typhoid fever, particularly after perforation, and *intestinal tuberculosis* may also be confused with perityphlitis. When complications exist, intestinal tuberculosis can be differentiated with difficulty from perityphlitis. Borchardt[10] reports a case of this kind.

The patient entered the hospital with a pleuritic effusion and diarrhœa. Tuberculosis was diagnosed. During an illness of three weeks, symptoms pointing to the appendix were so mild that they were overlooked. Before death peritonitis occurred, which was shown by the autopsy to have originated from a perforated appendix.

Where the local symptoms are obscure, the pain not well localized, and the fever curve of a continuous type, we must think of *typhoid fever*. All the characteristic symptoms of typhoid must naturally be considered, and the Gruber-Widal serum test made.* The differentiation between typhoid fever and perforative peritonitis may be very difficult, especially in recent cases with obscure symptoms.

Septic (cryptogenetic) forms of appendicitis may also give rise to diagnostic errors. As shown by the cases of Heubner[49] and Karewski[22], the symptoms are generally not sufficiently well defined, and the course of the disease so violent that the patient dies before all diagnostic possibilities can be excluded.

The cases of "pseudo-perityphlitis" (Nothnagel) and "appendicitis larvata" (Ewald) should also be mentioned. French observers (Talamon[26] and Rendu[50]) have described cases of hysterical peritoneal irritation which very closely resemble appendicitis, and which have been operated on. Nothnagel[51] has recently described a similar clinical picture under the name of "pseudo-perityphlitis." Under the name of "appendicitis larvata," † Ewald[52] has described a train of symptoms in which (as proved by subsequent operations), despite apparent hysteria, distinct changes occur in the appendix.

Complications

These are not infrequent. They may obscure the clinical picture, and, after the patients have passed through the actual attack, often cause death.

Bossard aptly compares the inflamed appendix to a bomb which may explode at any moment. The comparison is still further true in that it indicates the many different directions in which such a bomb may burst.

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* [The blood must also be examined for leucocytosis, which is absent in typhoid fever and present in appendicitis and suppuration.—Tr.]

† As correctly stated by many authorities (Gussenbauer, J. Israel, and Senator), the designation "appendicitis larvata" may easily produce misunderstanding, because it contradicts the fact that all the clinical data are present.

All complications arise in two ways: by means of thrombosis and embolism, or by extension of the inflammatory process by contiguity. The latter is decidedly the more frequent. As we have already seen, the perityphlitic process spreads most rapidly downward (forming pelvic and vaginal abscesses). It may also spread posteriorly (causing lumbar abscesses, vertebral abscesses, etc.), toward the diaphragm (subphrenic abscesses), toward the anterior abdominal wall, the hollow viscera, and even into the thorax. If rupture through the abdominal or thoracic wall takes place, a fistula may result. It is not our purpose to discuss all the clinical symptoms arising from these complications. The resultant conditions are understood with difficulty when weeks or months intervene between the primary disease and the secondary complications[53], especially when the perityphlitic attack itself runs an exceedingly mild course.

Gerhardt[54] in particular has called attention to the frequency of *pleurisy* as a complication of appendicitis. He has observed it in no less than forty-eight per cent of all cases of appendicitis. In the great majority (forty-two out of fifty) the pleurisy was on the right side, in seven cases on both sides, and only once on the left side. The pleurisy was generally serous; in a small number there was dry pleurisy. These cases are most probably explicable by the fact that the inflammatory process affects the retrocæcal connective tissue; from here the process continues upward through the lymph spaces of the retroperitoneal cellular tissue, advancing through the diaphragm to the right pleura.

The second class of complications originate through thrombosis of the appendicular vein. Thence the thrombotic particles are swept into the blood stream and reach a branch of the portal vein. Pylephlebitis and multiple liver abscesses develop, and, as observed by Gendron[55], an abscess may rupture through the diaphragm, causing suppurative pleurisy and pericarditis. Terrillon[56] has directed attention to purulent pleurisy as a comparatively frequent complication of appendicitis. Thrombi may become loosened from some of the branches of the inferior vena cava and be carried as emboli to the heart and lungs.

Thrombosis of the iliac or femoral vein is a rare complication. These thrombi originate through direct extension of the inflammatory process to the large venous vessels, or as the result of stasis following compression. Under these conditions, as shown by Fowler, fatal hæmorrhages may occur. Arterial thrombosis is very rare. Körte[10] has reported one case. Bull and Fowler[57] have published cases of rupture of abscesses into the iliac and femoral arteries. Most of these conditions can be diagnosticated only on the autopsy table. The cases of suppurative hepatitis, pleurisy, and pericarditis, already mentioned, may be recognised during life, but we are rarely able to establish the relation between them and the causative appendicitis.

An important complication of appendicitis, to which attention was called by Rotter[32], is chronic intestinal obstruction by angular kinking of the intestine in consequence of adhesions. Two cases were cured by division of the adhesions. A case of this kind, complicated by numerous abscesses, was observed and brilliantly diagnosticated *intra vitam* by Nothnagel[58].

The infrequent complication of pregnancy with perityphlitis will be briefly discussed. Gynecologists (Abrahams, Mundé, Hlawacek, Treub, McArthur, Marx, E. Fränkel[59], and others) teach that appendicitis during pregnancy is generally a very serious complication. Fowler claims that this complication always leads to abortion, miscarriage, and death. E. Fränkel regards this as too pessimistic. As the result of his own observations and a study of the literature of the subject, he has demonstrated that the gravity of this condition depends upon the variety and severity of the appendicitis. In mild cases the process may heal and the pregnancy run its natural course; in severe cases, localized or general peritonitis will produce abortion and generally the death of the mother.* The puerperal period may also be endangered by appendicitis. According to Fränkel, there are three possibilities: 1. In consequence of uterine contraction there is a break in the continuity of the periappendicular abscess wall, with subsequent rupture into the free peritoneal cavity and general peritonitis. 2. Fresh invasion of a former inflammatory area by the bacterium coli. This invasion may produce peritonitis as well as puerperal infection of the uterus. 3. Parametritis may develop from extension of the appendiceal process to the vessels coursing in the appendiculo-ovarian ligament (Clado and Durand), or in the retrocæcal tissue. In a case of probable induced abortion which came under my observation there occurred a severe appendicitis, which was cured by operation.

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* [Successful operations for appendicitis during pregnancy with subsequent delivery at term have been reported by Kraft[18], McCosh[19], Johnson[60], Gerster[61] (two cases), and others. An instance of recovery without operation and subsequent delivery at term has been published by Bayley[62].—Tr.]

TREATMENT

Typhlitis

I believe that, therapeutically as well as diagnostically, typhlitis should be considered apart from appendicitis. The treatment of these two affections is so entirely different that a separate description is necessary. We have previously (page 431) discussed the different forms of development of typhlitis. Most of these are mainly of surgical interest, and their treatment is practically that of appendicitis, to which we therefore refer the reader. Medical practitioners are principally interested in stercoral typhlitis. If the physician agrees with us that, although very rare, stercoral typhlitis does occur, the therapeutic methods to be used become obvious.

The main treatment consists in the removal of the impacted fæces. We should employ therapeutic methods which even in appendicitis do no harm, for it is impossible to always exclude the latter disease. As already mentioned (page 189), *I am decidedly opposed to the administration of laxatives.* For the purpose of softening the fæces I use cleansing enemata of oil, or mixtures of castor oil, cod-liver oil, and soda. In severe cases intestinal irrigation (page 179) may be used. By these means we generally succeed in softening the inspissated fæcal masses.

Should satisfactory evacuation follow these procedures, the intestine will require rest. For the reasons often stated, I would warn against repeated enemata in the expectation of more thoroughly cleansing the cæcum. On the contrary, after the patient has had a movement the bowels should be constipated by opium suppositories (0.02 to 0.03 gms. [of the extract?]) or tincture of opium (twenty drops given once). Some benefit may also be derived from cold compresses. After three or four days, when the subjective pain and the sensitiveness of the cæcum have ceased and the patient's general condition is satisfactory, another enema may be given.

During the inflammatory stage the diet must be fluid, and gradually increased as the inflammation subsides and disappears. Subsequent treatment consists in preventing fæcal accumulations; whenever possible, this is to be accomplished by dietetic means only, aided perhaps by mild laxatives, or still better by enemata. In this connection we refer the reader to the chapter on Constipation.

There still remains an important question: Shall patients with this form of typhlitis remain in bed? Most decidedly they should. The patient is allowed out of bed only when three days have passed without pain, fever, or gastric disturbances, when the general condition is good, and there is no longer sensitiveness to pressure in the ileo-cæcal region. After he has left his bed the patient must be advised to take care of himself for several days.

Perityphlitis

(*a*) *Acute Perityphlitis*

A few observations regarding the possible prophylaxis of perityphlitis will not be out of place. As far as I know, Sahli, in his Congress Report, was the first to touch upon this subject. It was later taken up and discussed by Penzoldt[30], and more recently by Ewald[60]. Authors are fairly well agreed that, to prevent a relapse and to provide against an attack of appendicitis, constipation must be controlled. Where family predisposition toward appendicitis exists, I agree with these conclusions. As regards care to prevent the swallowing of seeds, fish and other bones, I consider these precautions theoretical rather than practical. The findings of surgeons in appendicitis operations do not justify such precautions. As previously mentioned, foreign bodies are found only in a small number of cases.

The treatment of perityphlitis requires some preliminary remarks, by which we hope to make our general standpoint more clear. There is no doubt that autopsies and operations have already produced a reaction in the treatment of appendicitis. This reaction will certainly become greater in the future.

Thanks to a large literary and statistical material bearing upon the subject, the condition of the appendix and its surroundings can, in the majority of cases, approximately at least, be determined by abdominal palpation. This distinct advance in diagnosis lends aim and direction to present therapeusis, and at the outset demands neither surgical nor internal therapy, but simply a plan which will always keep in mind the anatomical relations of the diseased processes and their influence upon the general system. This standpoint permits at times a surgical view of the case on the part of the medical practitioner, and *vice versa*. In the therapy of appendicitis internal and surgical treatment should not and cannot be opposed; but when internal measures do not suffice, surgical intervention should be an aid to them.

The *internal treatment*, which we shall now consider, must be based upon the following principles:

1. Absolute bodily rest.
2. Rest of the intestines.
3. Appropriate diet.

Absolute rest in bed is one of the oldest and most important therapeutic laws. From the moment appendicitis is diagnosticated the patient must take to bed, and not leave it until the attack is entirely over. Simple as are these regulations, they are frequently broken, generally by the patient, but sometimes by the physician. In his excellent treatise Rotter has reported a number of serious results from non-observance of these simple rules.

The most important remedy in perityphlitis is opium, which was first employed by Volz. It has a very pronounced immobilizing action on the intestines, reducing their movements and reflex irritability to a minimum. In this manner salûtary adhesions may form, the peritonitis become circumscribed, and, according to Sahli, the shock of the peritonitis be lessened. Opium has a favourable effect upon vomiting, loss of sleep, muscular irritability, and, according to Penzoldt, it also lessens the thirst. Finally, I would call attention to the little-known diuretic action of opium, which, in view of intestinal decomposition, is not without importance. In the General Division (page 195) we have already given the underlying principles of the opium treatment of appendicitis; we shall here briefly repeat them.

In the first days of appendicitis, when pain, fever, and an increasing tumour are the most prominent symptoms, opium is especially appropriate. It should be administered systematically (tinct. opii, gtt. xx, every three hours; or ext. opii, 0.03 gm., t. i. d.). Suppositories of opium, each containing 0.05 gm. ext. opii, to be used t. i. d., are also applicable, especially where internal administration causes nausea or vomiting. Provided a good and active preparation be at hand, the same directions apply to opium given subcutaneously (ext. opii, aquos. sterilizat., 0.3 gms. in 10.0 c. cm.—dose, a Pravaz syringeful t. i. d.).

In opium therapy the main rule must be *avoidance of its lavish use.* When the process has reached or passed its highest point and defervescence begins, opium is to be discarded. I would especially warn against giving opium during convalescence. There is no apparent reason for continuing the drug, and it may produce intestinal paresis, from which the patients often suffer for the remainder of their lives, particularly, as is so often the case, if there had been a tendency to constipation.

Opium is indicated not only in the beginning of the disease, but also in suppurative and perforative appendicitis and in marked diffuse peritonitis. In these instances the doses must be increased until the pulse is slow and full, the general condition satisfactory, and the facies composed (page 195). In beginning collapse and in sepsis opium is without effect; in fact, it must be changed for the excitant class of remedies.

Some authors prefer morphin (subcutaneously) to opium. The advantages of opium are quite evident. The treatment of appendicitis with preparations of belladonna, which has also been recommended, has not yet been sufficiently tested. Ferrand[61] prefers belladonna to opium; he claims it has all the advantages and none of the disadvantages of opium (suppression of intestinal secretion, fæcal accumulations, increase of putrefaction). The few cases I have treated with belladonna are not sufficient for me to express an opinion of its effect.

The contraindication to laxatives of all kinds follows directly from the principle of absolute intestinal rest. In fact, there is seldom occasion for a laxative. If such necessity should arise, an oil enema is by far the most appropriate remedy; in these cases, as Penzoldt quite correctly recommends, the physician should give the rectal injection himself.

It need not be emphasized that the greatest possible precautions are required during the act of defecation.

Ice applications [in the form of the ice bag or the Leiter coil] to the ileo-cæcal region constitute a further immobilizing agent, for the patient is then forced to lie absolutely quiet on his back. The ice may also lessen the pain. We need not fear peristalsis from the cold applications, for daily experience has shown that this, in view of the powerful inhibitory action of the opium, is scarcely to be considered.

Ice is indicated as long as inflammatory symptoms continue and no fluctuating abscess has formed. In the latter instance ice is, to say the least, superfluous. The physician is often asked whether ice applications are to be continued during the night. This question cannot be answered generically. If sleep be thereby hindered, the ice may be removed and cold applications instead applied; otherwise there is no objection to the continuation of the ice during the night.

As regards diet, the principle of greatest possible intestinal rest also applies. This finds its extremest exemplification in absolute starvation during the stage of inflammation. I do not deny the theoretical justification of the absolute withdrawal of food, but, as already stated (page 154), I believe that it is too severe a measure. It is justifiable only in an etiologically obscure case of peritonitis or of intestinal obstruction with fæcal vomiting. In these instances, subcutaneous injections of salt or sugar solutions are the only means of subsidiary nourishment, but, with Penzoldt and Ewald, and as opposed to Treves, I do not, for obvious reasons, consider nourishing enemata indicated.

The most important dietetic details have been described in the General Division; we can scarcely add to that description.

Besides treatment of the perityphlitic attack, many cases require appropriate after-treatment or observation. Regulation of the bowels in particular demands attention. In this connection we refer to the recommendations given in the chapter on Chronic Constipation. After the acute attack has passed, inflammatory adhesions or more or less exudations may remain; the question then arises how these may be best removed. In recent cases I think it wisest to treat these exudations expectantly, and to advise rest and general bodily care. If the swelling persists, artificial or natural saline or mud baths are often beneficial, or even curative.

Massage is also often advised for the exudations following perityphlitis. We have stated in the General Division that massage is to be used only after the inflammatory symptoms have run their course, and must be practiced only by a *physician* experienced in this field of work. Other authors (e. g., Nothnagel) advise against massage.

The question regarding gymnastics and sports must be carefully decided. This question is the more apt to arise because appendicitis usually occurs at an age when exercise is an important factor. May a military officer ride, a gymnast exercise, a bicyclist ride, an oarsman row? These questions must be answered individually and with the greatest reserve. Under all circumstances, and for at least several months, exercises like the above should be prohibited. If no relapse occurs and no untoward symptoms set in, we may tentatively allow the patient to follow his special sport. Mountain climbing, either as a vocation or as a pastime, requires the greatest caution, and should not be extensively attempted for at least six months after the attack.

We have spoken of the use of baths, particularly of saline and mud baths, in the after-treatment, especially where there is constipation. These may be combined with water cures (Kissingen, Homburg, Marienbad, Carlsbad, Tarasp, Franzensbad, Rohitsch, Elster, etc.). [For corresponding American mineral springs and wells, see page 161.—Tr.] (Compare also the chapter on Hydrotherapeutics in the General Division.)

Operative Treatment

In view of the newness of the operative treatment of appendicitis, and of the customary separation of internal and surgical therapy, it is quite difficult for the internal practitioner to express an opinion concerning the indications for and the significance of operation. Our judgment will therefore depend upon the experience of surgeons who are acquainted with the results that can be obtained by internal treatment.

At every one of the numerous discussions within recent years there has arisen the preliminary question: How do the results of internal treatment compare with those of surgery? Sahli, Renvers, Kleinwächter, Rotter, Curschmann, Aufrecht, and others, have brought forward a very impressive material to show the curative results of conservative treatment. They have thus doubtlessly strengthened the cause of conservatism, and have contributed much toward preventing too radical surgical measures.

We quote Sahli's statistics, because they are very large and therefore most trustworthy. Sahli[1] collected the entire material of Swiss physicians, and thus gathered 7,213 cases. Of these, 473 were operated on, with a mortality of 21 per cent; 6,740 were treated conservatively, with a mortality of 8.8 per cent. Relapses occurred in 20.8 per cent. The figures of other medical practitioners and surgeons only partly agree with these. For instance, Kleinwächter gives a mortality of 7 per cent; Curschmann and Aufrecht of 4 to 5 per cent; Rotter, 8.9 per cent; Renvers only 3 per cent. The same is true of surgical statistics, which give a varying mortality between 9.6 per cent (Murphy) and 24 per cent (Richardson), the average being about 15 per cent. It is possible that the mortality will be lowered by increased experience, early operation, etc., but a mortality of 5 to 8 per cent will exist in appendicitis operations, no matter how timely and successfully the operation is performed (Rotter).

It is useless to compare the mortality rates of internal and surgical treatment. We might at most compare the several groups of appendicitis with each other (simple appendicitis, suppurative, perforative, with or without diffuse peritonitis, etc.). Aside from this consideration, as recently emphasized by Borchardt, the value of statistics in determining the good obtained from one or the other method of treatment is extremely doubtful.* There are too many incommensurable quantities to be considered, which in some manner must lead to a false conclusion. After all, the comparison of hospital death rates would seem the most reliable. Even here great differences exist. Thus, as the result of internal treatment in the St. Hedwig Hospital [Berlin], in 213 cases of appendicitis, Rotter gives the low mortality of 8.9 per cent. In the internal division of the Urban Hospital [Berlin] the mortality of appendicitis (132 cases) was about 12 per cent (Borchardt[10]), but of the 16 that died, about 14 were admitted with an inoperable general peritonitis. These statistics speak for themselves, and demonstrate that even the cases which come under the observation of one man are subject to many accidental variations. From this it follows that special rules for individual cases cannot be laid down, but that only underlying general principles can be given.

For the purpose of clearness we employ the usual subdivisions of appendicitis into simple catarrhal, suppurative, and perforative. We shall later, from the surgical standpoint, discuss chronic perityphlitis.

Simple catarrhal appendicitis does not usually necessitate surgical interference. Sonnenburg believes that in these instances the perityphlitic *attack*, and not the perityphlitic process, is cured. There is not, however, any ground for such conclusion. Simple perityphlitis will always be a medical disease.

In *circumscribed suppurative appendicitis* we must distinguish between cases with and those without abscess. In the former group there exists a possible indication for operation. When the patient's general condition is good we may await the absorption of the exudate, which undoubtedly takes place in a large number of cases. At all events the operation is generally simple, without danger, and usually cures the suppurative process in a short time.

It is quite a different question whether the appendix itself should be removed in suppurative appendicitis. Surgeons hold contradictory opinions on this subject. As far as I can see, Sonnenburg is the only German surgeon who recommends radical extirpation in order to prevent relapses. Fowler and Murphy are of the same opinion. The majority of the other surgeons—Körte[10], Schede[62], Rotter[32], Mikulicz[63], etc.—consider that simple incision is the proper procedure, because the removal of the appendix is very dangerous and sometimes technically impracticable. In a case of Gerhardt's[64], after extirpation of the appendix there was a relapse. The appendix may be removed when the patient is not thereby endangered—i. e., when the abscess is very small and well encapsulated, and also in those cases where the appendix is not walled off by adhesions from the general peritoneal cavity.*

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* Compare also the brilliant discussion of O. Rosenbach regarding the value of statistics in diphtheria, in the Münch. med. Wochenschr., 1898, No. 27.

Experimental studies (Wieland[65], P. Grawitz[66]) have demonstrated that the peritoneum is capable of absorbing small quantities of pus. Furthermore, Renvers has seen cases in which the presence of pus was shown by exploratory puncture, cured by conservative treatment. Finally, during operations, inspissated pus, evidently from previous exudates (Kümmel, Körte, and others), is often found in and around the appendix.

In addition to the above, pus may be eliminated in two ways: (1) By self-drainage (Sahli)—that is, by the pus emptying through the ostium of the appendix itself; or (2) by perforation of an extra-appendicular abscess. Pus can only rarely be discovered in the evacuations of the patients, but this fact does not speak against the theory of self-drainage, since pus in the stool soon undergoes changes which may make its recognition impossible (Sahli). On the other hand, extension of the suppurative process with diffuse peritonitis is quite frequent. These cases are often cured by surgical, and only exceptionally by conservative, methods. The question is still further complicated by the fact that, according to Rotter, even with general peritonitis the inflammatory process may become localized, lead to the formation of an abscess, and therefore heal spontaneously or after simple incision.

These complicated conditions make the decision regarding the time for surgical intervention one of the most difficult and responsible tasks of the physician. Should we operate while there is still hope that the process will heal under conservative treatment? How long should conservative treatment be tried? We must not forget the deceptive similarity between convalescence and danger. What shall determine the proper procedure in these cases: the unfavourable result of internal treatment, or the successful results of surgery? Not a few cases have been reported in which, both under conservative and surgical treatment, the disease unexpectedly took a favourable or an unfavourable termination.

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* [The operator will have to be guided by the condition present. Among American surgeons, Deaver[83] and Morton[84] favour the removal of the appendix in every instance. McBurney[85], Bull[86], Senn[87], Murphy[88], Mynter[89], Fenger[90], Fowler[34], and almost all other noted surgeons advocate a careful search for and removal of the appendix when the patient's general condition permits of reasonable delay, and when the location and extirpation of the appendix do not necessitate dangerous dissection and endanger the continuity of the abscess wall.—Tr.]

As a general principle for these cases, we would lay down Rotter's rules regarding the course of the temperature. He says: "If, despite proper internal treatment, the fever shows no tendency to subside, or rises after the third day, or if, after a slight remission, the temperature after the fifth day reaches 39° C. or over, operation should not be delayed." In such cases the patient's general condition will usually be disturbed, and the seriousness of the condition will be indicated by the vomiting, the frequent, soft, irregular pulse, tympanites, great bodily weakness, sleeplessness, marked sensitiveness to pressure in the ileo-cæcal region, and singultus. Rotter also says that cases which at first run a favourable course, but which after a number of days again have fever, should also be operated on. In all these instances an abscess is present, and if not incised may lead to serious complications.

Those cases which immediately, or after twenty-four to forty-eight hours, have signs of diffuse septic peritonitis, are undoubtedly surgical, and the sooner they are placed under the surgeon's care the better. Here, as already mentioned, the great difficulty frequently lies in the diagnosis. The majority of these patients can scarcely stand narcosis, let alone operation; hence the surgical results are not very encouraging. Thus, despite timely operation, Rotter lost sixty-six per cent, Körte sixty-four per cent, and Sonnenburg[67] fifty-eight per cent of such cases. Every successful result must be regarded as a direct gain.

(*b*) *Chronic Perityphlitis*

Concerning this affection a proper understanding is beginning to exist between medicine and surgery. We have already studied the two forms of chronic appendicitis: chronic appendicitis in its narrower sense, and relapsing appendicitis. Under certain conditions both forms may be accompanied by severe symptoms, which disturb the patient and interfere with his ability to do work, or even endanger his existence.

We must, however, remember that great care and the use of appropriate remedies (baths, springs, and diet) will remove many of the patient's symptoms, and that relapses are often much milder and do not last as long as the primary attack. Finally, as Rotter[32] and Kümmell[68] have shown, relapses are much more frequent within the first year following the primary attack, decrease in frequency in the second and third years, and are very rare thereafter. The results of surgical treatment are exceptionally favourable; Kümmell[68], who has had the most experience in relapsing appendicitis, does not consider removal of the appendix more dangerous than an ordinary ovariotomy.

The social status and vocation of the patient are of great importance in deciding for or against operation. A patient so situated that he may take every possible care of himself need not decide upon an operation as rapidly as one who must work hard for a living.

Hence, the following indications for operative interference in chronic appendicitis may be laid down:

1. If, after an acute attack of appendicitis, severe and other disturbances (pain, sensitiveness, etc.) persist, operation is to be performed as soon as possible in those whose vocation necessitates work. In other cases operation is indicated only after other remedies have failed.

2. In relapsing appendicitis, especially among the working classes, operative interference is indicated when the attacks occur at short intervals and become more and more severe. If the interval between the primary and the next succeeding attack is more than three years, operation may be delayed or advised against.

Borchardt gives two further indications which, for the purpose of completeness, we here repeat:

(*a*) In women, operation is indicated when the adnexa are affected by the inflammatory process. Early operation may prevent infection of the adnexa of the left side.

(*b*) Operation is indicated when, as a consequence of adhesions between the appendix and the female adnexa, severe symptoms occur during pregnancy, which tend to produce abortion or miscarriage.

Finally a few remarks on the operative treatment of *tubercular appendicitis* are in place. We have previously stated that, according to literature, this class of cases concerns individuals with a tubercular constitution in whom the appendicitis is only a complication. In my opinion, operation in these instances is as little indicated as in a tubercular kidney with marked pulmonary phthisis. The few cases which were operated (Körte and Sonnenburg) died in a short time. Borchardt reports two cases of tuberculosis of the cæcum with fistulæ in the ileo-cæcal region. Both patients died soon after operation.

The prognosis after operative treatment of *actinomycotic appendicitis* is also unfavourable. Of the twelve cases reported in Sonnenburg's monograph (to which we add another case recently reported by Karewski[22]) only one was cured.

In conclusion, a few remarks on the relation between the physician and the surgeon appears to me appropriate. We consider it very desirable that, wherever possible, in every case of apparently severe appendicitis a surgeon shall be immediately consulted. It is not necessary to operate at once, but the case is to be observed and studied by both practitioners, and the proper time for surgical interference watched for. In public hospitals this is readily accomplished, but in private practice it should be followed to a greater extent. The surgeon who in a given case decides for conservative measures instead of for operation, will not lose, but gain, in reputation.

[The preceding chapter describes very fully the modern continental view of appendicitis, particularly from the standpoint of the general practitioner. Whether from climatic, racial, dietetic, or other influences, the type of the disease in Europe is a far milder one than in the United States, or whether, because less prevalent, its gravity is not so fully appreciated, certain it is that the medical profession abroad regard appendicitis in a far more sanguine light than we do in this country. It appears therefore in place to summarize the American ideas of appendicitis as gathered from literature and personal experience.

Within recent times no other affection has been the subject of so much discussion, demonstration, study, and writing. In this country, appendicitis is of such frequency that almost every layman is acquainted with its manifestations and dangers. Fitz's[91] masterly monograph taught us the proper significance of, and gave the impetus to, the further study of ileo-cæcal inflammations. The brilliant operative results of Morton[84], Sands[92], McBurney[93], Weir[94], and others demonstrated practically what Fitz theoretically taught, viz., the possibilities of surgery in this domain. Rapidly experiences multiplied, definition in diagnosis, prognosis, and treatment became more exact, and to-day the clinical phases of the disease are far better understood. We have come to regard appendicitis essentially as a surgical condition, and one which must therefore be treated upon strictly surgical principles. It is everywhere recognised by general practitioners that each case of appendicitis ought (at least in large cities) to be treated by, or in conjunction with, a competent surgeon.[*] This fact is also understood by our general public; and once the diagnosis is made or suspected, the possibility of an immediate or future operation is entertained. In all our hospitals patients with appendicitis are assigned to the surgical division.[†]

The terms typhlitis, cæcitis, iliac abscess, pericæcal abscess, and perityphlitis, though still mentioned in text-books and monographs have been completely dropped in practice. That primary inflammatory (suppurative) conditions of the cæcum may occur is not denied, but if they do occur they must be exceedingly rare, and clinically indistinguishable from appendicular processes;[‡] therefore no practical value attaches to their separate consideration. Clinically, at least, the diagnosis " appendicular (vermicular) colic " is never made in this country, for that condition is regarded as identical with a mild form of catarrhal appendicitis (Hartley[77], Fowler[#]).

For the reasons stated at length in the main body of the present chapter, and which, together with a few additional facts, shall now be briefly recapitulated, appendicitis differs essentially from other inflammatory and ulcerative processes of the intestines.

The vermiform appendix in man, being in an evolutionary state, has a natural tendency toward obliteration; hence it can offer but a feeble resistance to deleterious influences. Owing to its very rich lymphatic structure, excessive secretion readily results from irritation. The disproportion between the length and the diameter of its lumen, the scarcity of contractile muscle fibres, the presence of fæcal concretions and of Gerlach's valve, and of other physiological strictures, and the pendent position of the organ, favour the stagnation of this excessive secretion. As a result, there is increased pressure within the appendix. Concretions and other foreign bodies produce erosions and ulcerations of the wall. The nutrient vessels being terminal branches, there is no provision for the establishment of compensatory anastomosis, the circulation is readily embarrassed, and, unless the stagnation within the lumen is relieved, gangrene results. The presence of bacteria adds an infectious element to the process, and may lead to the formation of an abscess within the appendix (empyema), or (with or without perforation) to suppurative processes in the surrounding parts. General sepsis may also occur.

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[*] [" It must be confessed that, according to our present views, appendicitis is a surgical rather than a medical affection, particularly from the standpoint of treatment " (Anders[95]).]

[" The disease is, properly speaking, a surgical one " (Lockwood[96]).]

[" In the majority of instances appendicitis is a surgical affection," writes Pepper[97], one of the strongest advocates of the opium treatment.]

[†] [Osler[98] remarks: " So impressed am I by the fact that we physicians lose lives by temporizing with certain cases of appendicitis, that I prefer, in hospital work, to have the suspected cases admitted directly to the surgical side."]

[‡] [According to McBurney, 99 per cent of all typhlitic abscesses are of appendicular origin.]

[#] [Loc. cit., p. 45.]

Usually the changes involve the entire appendix. Hartley[77] states that some appendices which he had removed in the first twenty-four to forty-eight or even seventy-two hours of the disease, showed changes only in the mucosa and submucosa. If the serous coat becomes affected, a local peritonitis, with the formation of more or less extensive adhesions, results. "These adhesions may repeatedly form an efficient protective wall, but often they are powerless to prevent the further spread of a purulent peritonitis. This is the most important phase of the pathology of appendicitis, and is a condition which we will never be able to overcome" (Stein[99]). The existence of such a condition is a strong argument against waiting for an abscess to become absorbed, or against delaying the operation until firm adhesions have formed. In very acute cases the infective process may spread so rapidly that there is no time for adhesions to form, and an acute general peritonitis supervenes.

In addition to empyema, ulceration, and perforation, the appendix may be the seat of other pathological changes, particularly where there have been repeated attacks. Strictures and cystic conditions may develop, the lumen may be obliterated, or the wall of the appendix become thickened and indurated. Sometimes the entire appendix is embedded in a mass of adhesions, and loses its original appearance and character.

No new facts relative to the *conservative treatment* can be added to those described on page 458. It is well, perhaps, to briefly define the position assumed in this country in regard to opium and cathartics.

As in other countries, views differ regarding the propriety of prescribing opium and its alkaloids in appendicitis. In private practice there is a constantly increasing tendency to limit their employment. Surgeons, as a rule, condemn their administration, at least until the diagnosis has been made or operation decided upon. Even then opium should be used in minimum amounts, just sufficient to relieve pain. It has been stated that a pain unrelieved by an ice bag, etc., and severe enough to require large doses of opium, constitutes in itself a sufficient indication for operation (Wiener[100]). The objections to opium are: (*a*) That it masks the symptoms and produces a false euphoria, rendering it impossible to properly estimate the attack; (*b*) that it favours the development of a tympanitis which we may be unable to differentiate from that of a beginning peritonitis (Wiener); and (*c*) that in large doses it is apt to induce an intestinal paralysis. The points in favour of its use have already been mentioned (page 458). Pepper[97] considers opium in full doses as "the great standby," and that it has greatly lessened the mortality from appendicitis. Einhorn[101] considers opium the remedy *par excellence*. Deaver[102] permits its use for the relief of pain, but only after a purgative action has been obtained; he considers the local use of ice much better than the administration of opium. Lockwood[96] also would give opium, but not to the extent of semi-narcotism.

The use of cathartics is still a disputed question. Recently salines were extensively employed and recommended by surgeons. Deaver[102] claims never to have seen any harm from catharsis. He certainly does not voice the general opinion when he says: "Purgatives are capable of doing much more good under these circumstances than any other class of drugs. . . . I am positive, after considerable experience, that the good from purging will overbalance by far the harm done by active peristalsis. The writers who oppose the use of these drugs are evidently limited in their experience with the disease, otherwise they would not so believe." Tiffany[103] also favours free purging. In general, both medical and surgical authorities condemn their use during an acute attack (Osler[98], McBurney[85], Einhorn[101], McNutt[104], Mynter[89], Pepper[97], etc.). Tyson[105] opposes their employment in advanced cases, but thinks their early administration in mild or moderate cases may clear up the diagnosis, or, by depletion of the circulation, diminish the danger of peritonitis.

Against the use of cathartics it is urged that they are not needed, since the cæcum is rarely filled with fæcal accumulations; that cathartics tend to increase nausea and general unrest; and that by exciting peristalsis they prevent the formation of fresh adhesions, and break up those already formed, but not yet firm. Finally, it must not be lost sight of that, even in the very earliest stages of appendicitis, we can never tell how near the appendix is to perforation. Hence, in general, it is best to defer the administration of purgatives until after operation, or until the attack has passed off. If it be necessary to empty the lower bowel earlier, an enema will answer very well.

What are the recognised indications for operation? In endeavouring to answer this question the writer has consulted the publications of recognised American authorities,* and has also drawn upon personal experience in hospital and private practice. He finds that there has been a considerable change of opinion since the clinical and pathological manifestations of appendicitis have received more direct attention. In the earlier days it was universally recommended to wait until an abscess had formed. The discovery and publication of McBurney's point[106] was a most decided advance in the early diagnosis of acute appendicitis. Taught by sad experiences, the American profession has come to appreciate the dangers which attend postponement of operation, and, emboldened by the success of modern surgical methods, has learned to be more radical in its treatment of appendicitis.

We recognise that owing to anatomical peculiarities, an appendix once the seat of a more than very slight inflammation will never return to its normal state, and that this predisposes the organ to fresh attacks. We believe that with each fresh attack the pathological state of the appendix is aggravated. There are no specific internal remedies for the cure of appendicitis. Treated conservatively, mild cases often recover with a *restitutio ad integrum*, or with no further changes than strictures of the lumen; severer cases may be attended by any of the processes already mentioned. Exceptionally, an extra appendicular abscess ruptures externally or into a hollow abdominal viscus (bladder, cæcum, rectum, etc.), and relief or spontaneous cure follows.†

The abscess has occasionally perforated the diaphragm and discharged through the lung. The danger and inconvenience of such conditions are too apparent to call for any comment.

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* [See Literature at the end of this chapter.]

† [Lloyd[107] reports a very interesting and instructive instance of acute appendicitis occurring in a man in whom years before a peri-appendicular abscess had ruptured and discharged into the rectum.]

In the vast majority of cases we are unable, without laparotomy, to determine the condition in the ileo-cæcal region. We cannot tell how near the appendix is to perforation; whether there are any adhesions, and, if so, how firm; whether there will be any further attacks, etc. To guide us in our disposal of a case, we must appeal to our experience and the results of pathological research.

With but few exceptions the profession have therefore come to regard appendicitis as a surgical affection. This applies as well to the simple catarrhal as to the suppurative and other severer forms. If, then, during a slight or moderately severe acute attack we resort to rest in the recumbent position, an ice bag, restricted diet, and other conservative measures, in the endeavour to tide the patient over the attack, we are none the less fully alive to the possibility of an immediate or the probability (almost certainty) of a future operation. We watch our cases very carefully, and are prepared for immediate surgical interference should indication arise. Experience has taught that the period of quiescence between attacks offers the best chances for operative success. The patient or his friends are instructed concerning the gravity of the situation, and such details are explained to them as are necessary for their clearer judgment. For with the family, after all, will rest the consent to operate.

Contraindications to Operation.—Besides the withholding of the consent of the patient or those responsible for him, operation may be impossible or inadvisable for other reasons. Such would be inadequate surroundings, failure to obtain proper assistance, surgical inexperience of the medical attendant, too far advanced condition of the case (sepsis, extreme weakness, moribund state, etc.) coincidence of other serious disease, etc.

In the absence of these adverse circumstances the following are the generally accepted *indications for operation:* *

I. Interval operation.†

(*a*) In mild cases after two or more attacks.

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* [The following indications apply only to patients residing in large cities or otherwise accessible to immediate surgical interference. For those who travel much, who live in the country, or who must perform severe physical labour, etc., it is best to remove the appendix during or after a first attack.]

† [It is best to wait about two to four weeks after even mild attacks, until the inflammation has become quiescent. The mortality under such circumstances, even in difficult and unfavourable cases, is, in the hands of a good operator, 1 per cent or 2 per cent (McBurney).]

Willy Meyer[108]* advises the radical operation after recovery from any attack of appendicitis, mild or severe. Deaver[83], Mynter[89], and a few other surgeons, favour immediate operation in all cases of appendicitis as soon as the diagnosis is made.

(*b*) After recovery from an attack of ordinary or more than ordinary severity.

Here general practitioners and surgeons are almost unanimously agreed that it would be assuming too great a risk to expose the patient to the dangers of a repeated attack.

II. Immediate operation.

1. In cases of ordinary severity with sharply defined symptoms.

(*a*) Whenever there is a tumour present in the ileo-cæcal region.

(*b*) Whenever there is sudden or progressive increase in the gravity of the symptoms.

(*c*) When, after thirty-six to forty-eight hours, the case does not show any tendency toward improvement, but the condition remains stationary.†

(*d*) Whenever there is any doubt as to the existing condition and the patient's improvement most authorities advise immediate operation. In this instance timely operation is better than the uncertainty and dangers which attend delay.‡

2. In all the severer forms of appendicitis—i. e., those in which the symptoms point to pus in or about the appendix (with or without peritonitis), to perforation, or to severe systemic infection—we cannot operate too early. It is wrong to delay and attempt to determine the pathological conditions present, for we will rarely arrive at more than a probable diagnosis, and every delay may cost the patient his life. In all cases of acute appendicitis the earlier the operation the easier its performance, the better the condition of the patient, and the more certain are the chances of success.—Tr.]

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* [Willy Meyer reminds us that the first attack is really not the first pathological symptom, but rather the "first explosion."]

† [Here one must be guided, in advising immediate or interval operation, by the existing circumstances, particularly by the general condition of the patient and the care with which the case can be watched.]

‡ [McBurney[85], one of our greatest authorities on appendicitis, says, in referring to this indication for operation: "No greater mistake can be made than to wait for very clearly defined signs of advanced and grave disease before deciding to operate. Operation, to be usually successful, must be done before grave disease is well pronounced."]

APPENDIX

Sigmoiditis and Pericolitis

(a) *Acute Sigmoiditis*

Under this name A. Mayor[69] has described a condition which consists in an inflammation of the sigmoid flexure and occasionally of the neighbouring peritoneum and cellular tissue ("iliac phlegmon"). Mayor believes that this affection is caused by fæcal impaction in the sigmoid or by irregular defecation. Both causes may, however, be absent. Fever may or may not be present. The region of the sigmoid is always indurated, swollen, and sensitive to pressure; suppuration and rupture into the intestine may occur. All the cases were marked by rapid convalescence. Mayor leaves it undecided whether the disease is produced by purely mechanical factors or by specific infection (e. g., erosions).

To illustrate the clinical picture, it seems best to present a brief analysis of the clinical histories reported by Mayor:

Case I.—A woman, thirty-two years old, who, excepting for constipation, had always been well. On examination of the region of the sigmoid flexure, there was felt a cylindrical swelling which was continuous with the descending colon above, and disappeared toward the pelvis. Rectal examination showed nothing abnormal. This condition developed without fever, and disappeared under the use of cataplasms.

Case II.—Boy, fourteen years old, became ill with fever, pain in the left side, and local symptoms similar to the above; after fever for several days the boy recovered.

Case III.—Boy, eleven years old, became ill with high fever; suppuration occurred, and the abscess ruptured into the intestine.

Case IV.—Physician, forty-two years old, sudden, severe, paroxysmal pains in the sigmoid region, accompanied by nausea; no constipation, no fever. The sigmoid flexure was indurated, swollen, and sensitive. Recovery after a few days.

In a case recently reported by Galliard,[70] there were severe general symptoms, with fever, and pain in the left iliac fossa. There was a tumour the size of an orange, which disappeared after three weeks.

In the absence of autopsies, certainty regarding the pathogenesis of the disease is scarcely possible. It seems very difficult to differentiate acute sigmoiditis from inflammation of the cæcum, or an appendix displaced to the left. Chronic enteritis with acute exacerbations may also be mistaken for sigmoiditis, especially since fever is not a necessary symptom of the latter. Finally, inflammation of the left female adnexa may also require diagnostic consideration.

Mayor deserves credit for having pointed out that inflammatory processes may occur in the left iliac fossa. Further study of these conditions, and the knowledge derived from operative procedures, will be required before we can say that there is proper justification for considering this affection as an inflammation of the sigmoid flexure.

The same *treatment* must be applied as in typhlitis: rest in bed, ice, or, when this is not well borne, warm applications, regulation of the bowels, and opium (internally, subcutaneously, or in suppositories) when the pain is severe.

(*b*) *Chronic Sigmoiditis*

By chronic sigmoiditis I mean an affection in which there is constant pain and sensitiveness to pressure in the region of the sigmoid flexure. Further symptoms are severe diarrhœa, accompanied by more or less mucus, or by attacks of constipation.

Von Leube[71] and Rosenheim[72] also report chronic inflammatory infiltration of the sigmoid flexure with a "smooth, regular, increased resistance." Since chronic sigmoiditis is but little known, I will briefly report two cases from my journal:

Case I.—Mrs. F. R., of Berlin, age thirty-nine. No hereditary disease; has had four severe labours and two abortions, produced, she says, by falling and jolting. Her present symptoms have lasted eleven years, and are ascribed by her to the first confinement, which was an instrumental delivery with complete tear of the perinæum. A recto-vaginal fistula remained, and caused the patient much annoyance. Three years later the fistula was operated on by Prof. Fritsch. Patient felt better for a few months after the operation, but the symptoms gradually returned, and at present they are at their greatest intensity. They consist of pains in left lower abdominal quadrant, flatus, and morning diarrhœa (two to four movements) with much mucus. Blood has never been found in the stools. Frequent tenesmus, which only ceases after injections of chamomile infusion. The diarrhœa alternates with normal stools for one to two days. On such days the patient feels much better. The other gastro-intestinal functions are absolutely normal. All treatment (oil enemata, opium, tannalbin, etc.) has been without effect up to the present time.

Status Præsens.—Pale, well-nourished woman. Organs of respiration and circulation normal.

Abdomen.—Many striæ, abdominal panniculus flabby (pendulous abdomen), splashing and succussion sounds in the gastric region. Lower border of the stomach (carbonic-acid inflation) reaches to the umbilicus.

The slightest *palpation* in the region of the sigmoid is extremely painful. As the examining finger passes upward the sensitiveness becomes less, and at the splenic flexure disappears entirely. No resistance to be felt. Rectal examination negative.

Urine.—Normal; no indicanuria.

Stools.—Two to three stools daily, of semisolid consistency, and mixed with viscid mucus. Microscopically nothing of importance, particularly no blood, pus, or amœbæ.

Stomach.—Motility normal, marked hyperacidity (0.28 per cent HCl).

During the clinical examination there were six to seven thin stools, accompanied by severe tenesmus. The treatment consisted in absolute rest in bed, astringent diet, hot poultices over the sigmoid flexure, and rectal irrigations with bismuth mixtures (bismuth, ½ ounce; water, 1 litre).

After four weeks of treatment patient showed considerable improvement; had one to two well-formed stools daily; painless defecation, and much diminished sensitiveness over sigmoid.

CASE II.—Mr. S., of W., thirty-two years old. In February, 1896, while in India, he had a severe attack of acute dysentery, which slowly improved after about four weeks. A sensitive area remained, however, in the left lower portion of the abdomen. Pain was particularly evident while riding, so as to necessitate the giving up of that exercise. After walking about for several hours the patient usually felt more or less severe pain in the above-mentioned area. Appetite good. Stools always show a tendency to diarrhœa. Examination reveals circumscribed sensitiveness and a feeling of light resistance over the sigmoid flexure. Everything else normal.

These cases demonstrate that local inflammation of the sigmoid does occur. In the first instance the etiology was very probably an infection originating in the recto-vaginal fistula; the second case very probably originated from the previous dysentery.

The *diagnosis* is made from the catarrh of the large bowel, and from the sharply defined sensitive area corresponding to the sigmoid flexure. There need not be a demonstrable resistance. Differentially, we must, in the first place, consider malignant neoplasms, and then diseases of the female adnexa. By careful examination we ought to be able to exclude both groups of diseases.

The treatment is the same as that of chronic catarrh of the large intestine.

(c) *Exudative Pericolitis; Primary Submucous, Circumscribed Colitis* (*Pal*)

Under the name of exudative pericolitis, Windscheid[73], of the Leipsic Clinic, first described a condition characterized by the development of acute exudative peritonitis about the ascending colon. This affection is distinguished from typhlitis and appendicitis in that the right iliac fossa is entirely free, and from tumour by the acute onset and constant fever. In the same year Eisenlohr[74] reported "a case of abscess behind the ascending colon." Since this is the only instance in which an autopsy was performed, its important points are here briefly recapitulated.

A ferryman, thirty-one years old, alcoholic, with cirrhosis of the liver, on March 31st became ill with chills, vomiting, diarrhœa, and marked abdominal distention. On examination, the right hypochondrium was found sensitive to pressure, and painful ; pulse frequent ; diarrhœa ; vomiting bilious but not feculent ; urine contains no albumin ; peptonuria found once.

March 24th. Fluctuation in the abdomen with disappearance of peritonitis ; later, ascites ; evening temperature 39° C.

Death on May 29th. Autopsy showed a small abscess cavity situated below the upper portion of the ascending colon and between the hepatico-colic ligament, the anterior surface of the kidney capsule, and the descending portion of the duodenum. This cavity contained a scanty amount of semisolid, inspissated, yellowish pus. The mass measured about ten centimetres vertically, and somewhat less horizontally. Its position corresponded to the mesentery of the upper portion of the ascending colon. The walls of the abscess cavity were tough, and thickened by connective tissue ; the peritoneum of the intestines, particularly that of the cæcum and appendix, showed no trace of previous inflammation. The entire mucous membrane presented no evidence of previous ulcers, cicatrizations, or infiltrations. The abscess was entirely outside of the intestinal wall.

In 1895 Fleiner[75] reported a case which he also described as a pericolitic exudate. J. Pal[76] recently published a series of such observations, in which he thoroughly discusses the question. Pal's cases include exudates about various portions of the large intestine, five cases of ascending colitis, one of colitis of the left or both flexures, one of colitis of the right flexure, and one of descending colitis. He considers colitis as a submucous infiltration which is developed from peculiar changes in the intestinal contents, and which either suppurates and ruptures or is absorbed.

I also am in a position to report a case of this kind which, after laparotomy, came to the autopsy table.

H. K., nineteen years old, student. As a child suffered from pertussis and frequent pulmonary and intestinal catarrhs. In 1889 tubercular knee and hip-joint disease, cured by extension and iodoform injections. After the chloroform narcosis then necessary, for the first time there developed vomiting, with colicky pains and marked sensitiveness in the left side of the abdomen. Cure after eight days. Then absolutely well for six years. On March 22d, 1897, another attack after drinking cold beer. Severe pain in the umbilical region, marked vomiting, and constipation. These attacks were repeated five times in four weeks, each time lasting one to three days. The last attack began April 20, 1897, with severe colicky pains to the left of the umbilicus, and radiating to the back. Vomiting and constipation ; no fever (?). When seen in consultation (April 23d), to the left of the umbilicus there was felt an intensely painful, incompressible resistance the size of an apple. Since the attendant physician has given the patient laxatives and irrigations, fæcal tumour can be absolutely excluded. Neoplasms also, because of the intense sensitiveness. The diagnosis of serous exudative pericolitis was therefore made.

Treatment.—Rest in bed, ice bag, opium *per rectum*, fluid diet. First stool four days after these regulations. Resistance can still be felt. Four days later, tumour can no longer be felt. May 5, 1897, tumour had entirely disappeared; no sensitiveness in the area above mentioned. General condition good. Course of waters at Kissingen in the summer of 1897 followed by favourable results. In autumn, 1898, new attacks at intervals of three to four weeks; at the last attack (end of October) there was absolute retention of stool and flatus. On October 31st, laparotomy performed by Professor Riedel, of Jena. The following facts are borrowed from his report of the case:

Incision shows the transverse colon moderately distended by gas; isolated white patches in the mesentery. To the left and above the cicatrized omentum is adherent to the tip of the spleen, which is very movable. The splenic flexure is extremely coiled, the individual coils being connected to one another by glistening, white, cicatricial tissue. Loosening of the mesenteric adhesions. Ether bronchitis; death from diffuse peritonitis.

In my opinion, these numerous scar tissue adhesions were the remnants of many previous attacks of exudative peritonitis.

Despite the small number of cases which have been observed, there is no doubt that, clinically, there do occur serous or purulent exudates without involvement of the appendix. Their etiology is difficult to determine. The explanation given by Pal is founded upon a case of Eisenlohr's, but since the entrance point for the development of the abscess is unknown, this case must be used only with the greatest caution for the establishment of a new disease.

Symptomatology and Diagnosis

According to Pal's description, the onset is sudden, with symptoms of inflammatory swelling of the large intestine, accompanied by fever, nausea, or vomiting; there soon develops a sensitive area, painful to pressure, and, in a few days, a palpable resistance. The resistance may rapidly increase in extent; at this stage the tumour is quite sensitive to pressure. When fever subsides the sensitiveness also disappears. The rest of the abdomen is slightly or not at all affected by the process.

The most frequent site of the disease is the hepatic, less frequently the splenic flexure. The process may develop in other segments of the large intestine, particularly in the ascending colon. When affecting the descending colon or the sigmoid flexure, the clinical picture is the same as that of sigmoiditis (Mayor). Occasionally, several intestinal segments may be simultaneously involved. In the beginning the clinical picture may impress one as that of a circumscribed peritonitis, but the rapidly developing cylindrical area of resistance points directly to the intestines as the origin of the lesion.

The fever is generally of short duration and may be overlooked, but when suppuration supervenes it may become quite marked. In the beginning the bowels may be normal or constipated. Frequently there is an accumulation of gas. There is often marked indicanuria.

DIFFERENTIAL DIAGNOSIS

Differentiation from typhlitis and perityphlitis is the first consideration, particularly when the process is localized on the right side in the neighbourhood of the cæcum. Pal places a certain value on palpation and percussion for the separation of the cæcum from the diseased colon. He, however, seems to undervalue the difficulties of an exact differential diagnosis of this disease from perityphlitis. When we consider the numerous variations in position of the cæcum, and the different localities of perityphlitic abscesses and exudates, error would appear unavoidable. When the colitis is situated in the vicinity of the right hypochondrium, cholelithiasis may have to be considered, and, under complicating conditions, the diagnosis may be very difficult. Perigastritis and perinephritis may also come into question.

TREATMENT

In general the treatment embodies the usual principles employed in all inflammatory processes of the intestine—rest in bed, ice, intestinal irrigations, and opiates when the pain is very severe. During the first few days fever diet should be given; in the following days the diet is that of appendicitis after the acute inflammatory symptoms have disappeared (see page 154). After the exudate has fully developed and acute symptoms have diminished, warm applications are in place. Pal particularly recommends hot flaxseed poultices. We may later attempt to aid absorption of the remainder of the exudate by massage, and iodin and mercurial ointments. When a fluctuating abscess develops, incision should not be delayed.

Repeated attacks of pericolitis or signs of chronic adhesive, intestinal inflammation may require surgical treatment, but at present the success of such operations is doubtful.

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CHAPTER XX

DISEASES OF THE RECTUM *

1. Proctitis

PROCTITIS is an inflammation of the mucous membrane of the rectum. It may be acute or chronic, circumscribed or diffuse, primary or secondary. Its causes are but little understood. Undoubtedly catarrh of the rectum may be caused by entozoa, particularly parasites of the large intestine (especially oxyuris vermicularis). The theory of an infectious proctitis in pruritus ani, hemorrhoids, foreign bodies, neoplasms, prolapsus ani, mechanical irritation, etc., is very plausible. The view constantly expressed, even in the most recent text-books and monographs, that there exists a connection between proctitis and the abuse of drastic purgatives, does not seem sufficiently proved. Among numerous observations, I have found but one instance of this kind. It is doubtful whether cooling of the pelvic region may produce proctitis.

The most frequent cause of primary proctitis is best sought for in the composition and character of the evacuations themselves. These may both chemically and mechanically, and under certain circumstances also bacteriologically, produce acute or chronic catarrh of the rectum. The most frequent causes of secondary proctitis are foreign bodies (fruit seeds, meat bones, fish bones, etc.), ulcerations, neoplasms, hemorrhoids (so-called mucous hemorrhoids), prolapsus ani, and rectal fistulæ. Infectious diseases (gonorrhœa, diphtheria, dysentery) are also looked upon as etiological factors.

As shown by recent investigations, *gonorrhœal* proctitis may occur both in men and women. In men it results only from sodomy. In women it is very frequently due to the carrying over of gonorrhœal secretions to the anal region, and, more rarely, to extension of inflammation from the Bartholinian glands, recto-vaginal fistulæ, etc. Jullien[1] and Baer[2] have recently investigated the existence and nature of rectal gonorrhœa. The latter investigator found this disease in thirty-eight per cent of all women infected with gonorrhœa, though rarely with any subjective symptoms. Gonococci are constantly found in these cases. Proctitis occurs frequently in women with extreme uterine displacements. Disease of the bladder and prostate may also produce proctitis. Buche[3] states that gout may also cause proctitis, but the connection between these two diseases has not been proved.

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* The scope of this work only allows of a description of those rectal diseases which are particularly interesting to the medical practitioner. Malformations of the rectum, wounds, and foreign bodies will therefore not be discussed. Rectal cancer is found in the chapter on Intestinal Cancer (page 318); the neuroses of the rectum are found among the Intestinal Neuroses.

SYMPTOMATOLOGY AND DIAGNOSIS

The symptoms of proctitis vary with the severity and extent of the process. In *acute* cases the inflammatory symptoms predominate; in the milder ones there is a feeling of fulness and pressure in the rectum. When the inflammation is severe the patients may complain of marked boring pain, accompanied by annoying tenesmus. The pain may be limited to the rectum, or may radiate toward the back, external genitals, bladder, and lower extremities. The movements are generally voluntarily repressed, and every effort at evacuation produces spasm of the sphincter and levator ani. If, after continued tenesmus, the patient finally has an evacuation, the latter is always small and is scarcely ever purely feculent, but is mainly bloody and muco-purulent.

In the severest cases the general health is usually involved. The pain keeps the patients in bed, appetite and sleep are markedly diminished, and from the very beginning fever is generally present. The finger introduced into the rectum feels the contracted sphincter, which allows further introduction only after very careful and gradual dilatation. The mucous membrane feels hot, œdematous, and swollen. Even when most carefully performed, digital exploration is very painful. On withdrawal, the finger is generally found smeared with blood or blood mixed with pus.

In *chronic proctitis* the symptoms are much less characteristic. There is a feeling of straining and pressure, which in the presence of impacted fæces may amount to tenesmus. Digital examination is less annoying to the patient. The mucous membrane feels swollen, soft, and velvety; it bleeds easily, and occasionally secretes pus. Sometimes the swollen solitary follicles may be felt as prominences the size of millet seeds. Defecation may be painful. The stools are often mixed with blood or bloody mucus; in isolated cases (e.g., rectal gonorrhœa) they may consist entirely of pus. In acute as well as in chronic proctitis, a paralysis of the sphincter may result from the marked and deep infiltration of the inflammatory process, so that there is a continual discharge of bloody pus or muco-pus.

Where the symptoms are well defined, the diagnosis of *acute* proctitis is usually easy and may generally be made without digital examination. For the diagnosis of the etiology digital exploration is necessary. It should be performed with the greatest caution. A very thin suppository of opium and cocain or eucain should be previously introduced. In place of this we may employ a collapsable tin tube to which a short piece of rubber tubing is attached. The latter is well oiled and passed into the rectum. The collapsable tube is then slowly squeezed and some of the ointment thus directly introduced into the rectum. The introduction of a speculum, especially without narcosis, is an unnecessary procedure. Fever, tenesmus, and bloody, mucoid, or purulent defecations with very little fæcal matter are also of diagnostic importance.

The diagnosis of *chronic* proctitis is best made by digital and speculum examination. Thus foreign bodies producing this affection can scarcely escape discovery. Under certain circumstances the diagnosis of the nature of the process may be very difficult. If ulcerations are present, we must consider especially the tubercular, syphilitic, gonorrhœal, and stercoral ulcers. These must be differentiated by clinical and, more particularly, by bacteriological examination (gonococci, tubercle bacilli).

If no sufficient explanation for the occurrence of the disease is found in the rectum itself, the etiology must be sought for by examination of the remaining pelvic organs—in men, the bladder; in women, the genitals.

TREATMENT

Depending upon circumstances, the treatment of *acute* proctitis should be either symptomatic or radical. The latter plan is applicable when foreign bodies, inspissated fæces, diseases of the neighbouring organs, etc., are the cause of the proctitis. Symptomatic treatment must take into account those basic principles which apply to acute inflammations of the intestinal mucous membrane—above all, absolute rest and immobilization of the rectum. The abdominal or lateral posture is often recommended for the relief of the pain. If constipation is not the cause of the lesion, it is advisable during the acute inflammatory stage to keep the bowels constipated by opiates, given either per mouth or rectum. It is also necessary to decrease the amount and kind of the nourishment taken—an easy task in view of the diminished appetite of these patients. When the inflammatory symptoms have subsided the bowels may be moved by castor oil or by enemata of olive oil. After this expectant treatment has been applied for eight or ten days, the symptoms, in the vast majority of cases, disappear. In complicated cases the process extends, the suppuration spreads to the periproctitic tissues, and a periproctitis results (see below).

Tenderness and tenesmus are generally well controlled by the above-mentioned narcotics. Leeches applied in the neighbourhood of the anus are also useful, and often curative; their action is increased by warm sitz baths.

Only after the acute symptoms have subsided should local treatment be begun, if at all. The utmost care must be exercised in the choice of our remedies. The most appropriate treatment is rectal irrigation with warm decoctions of chamomile or linseed, containing a few drops of laudanum. Where there is copious purulent secretion, we may try irrigations with very dilute solutions of nitrate of silver (0.5 to 1 gram in 1,000); if these cause severe reaction, they should be followed by an irrigation with a weak salt solution.

The management of *chronic* proctitis differs somewhat from the above. The first and leading principle is the recognition and treatment of the primary disease. Symptomatically, we must regulate the bowels, and keep the parts as clean as possible through copious rectal irrigations. It is immaterial whether for this latter purpose (of cleanliness) we use permanent rectal drainage with the apparatus proposed by Hofmokl (with which, moreover, there seems to have been no extensive experience), or the usual double-current catheters, or, as described in the General Division, a simple catheter with a T tube similar to that used in stomach lavage. According to my own observations, two, or at most three irrigations per day are sufficient. The permanent introduction of even a soft instrument is so uncomfortable that patients are seldom willing to undergo this procedure.

We have a large variety of antiseptics and astringents to choose from: alum, or its double salt, aceto-tartrate of aluminium (2 to 3 grams per 1,000), sulphate and sulpho-carbolate of zinc (1 to 2 grams per 1,000), tannic acid (3 to 5 grams per 1,000), nitrate of silver (0.5 to 1 gram per 1,000), etc. A change of solutions sometimes seems to produce a more rapid cure.

These irrigations are undoubtedly the most effectual of our therapeutic measures; we may, however, obtain favourable results with suppositories containing the above astringents. An ointment syringe has been recommended for the same purpose, but in view of the application of ointments by the collapsable tin tube (see page 484), which has given me the most satisfaction, an "ointment syringe" seems superfluous. We may in a measure conduce to cleanliness, and thus indirectly to the cure of the disease, by appropriate medicated sitz baths and by anal douches. In England, internal medication with balsam of copaiba, extract of cubebs, or oil of turpentine is highly recommended. I have not found it successful in my cases. The use of sea baths and other balneotherapeutic measures is practised by balneologists; but thus far no convincing proofs of their good effects have been adduced.

Some cases, especially resistant ulcerative and gonorrhœal proctitis, are extremely obstinate to all internal therapeutic measures.

2. Periproctitis

This is an inflammation of the loose cellular tissue about the rectum. Since this tissue is continuous with the fatty tissue which fills the ischio-rectal fossa on both sides of the termination of the rectum, between the levator ani and the sacral origin of the gluteus maximus muscles, the bulbous urethra and the perineal fascia, it is evident that local purulent processes may spread extensively. The etiological factors are the same as those already described under proctitis. Besides these, periproctitis may also, though now very rarely, occur after operations on the rectum which have not been sufficiently aseptic. Traumatism is also regarded as a cause of periproctitis, particularly since Cruveilhier has described a very marked case of this kind, resulting from a "fall on the gluteus." As far as I can learn, modern literature—which certainly contains abundant cases of accidents—does not present any instance similar to this.

The inflammatory process may be either acute or chronic, circumscribed or diffuse, the acute diffuse type being the most frequent.

From the seat of infection, the pus burrows through the cellular interstices and extends into the ischio-rectal fossa. The perineal fascia offers a slight barrier to the spread of the process, so that the anal region is generally affected later than the upper rectal segment, about which the pus may spread freely in all directions. Under these circumstances the abscess may rupture either externally or internally, and thus produce fistulæ (*q. v.*).

SYMPTOMATOLOGY AND DIAGNOSIS

The symptoms of periproctitis point directly to the seat of the lesion. In *acute* periproctitis the most persistent *subjective symptom* is the severe pain, and a feeling of fulness and tension in the anus and anal region. The pain becomes unbearable during defecation, so that patients voluntarily retard this act as much as possible. The disease generally begins with a marked chill, accompanied by high fever. When the process is very extensive—an occurrence occasionally found in cases improperly treated—the fever becomes septic in character. As might be expected, the general health soon suffers; the patients are considerably weakened by the fever, anorexia, and loss of sleep, and present the picture of an acute infectious disease.

The *objective* symptoms are usually very evident. There is a more or less hard, reddened, sensitive infiltration about the rectum. The introduction of the finger causes severe pain, and reveals a hot, swollen mucous membrane, which narrows the lumen of the rectum. Bimanual examination elicits fluctuation relatively early. In women a vaginal examination may also be made.

In *chronic* cases the symptoms are less defined. The abscess may rupture without the patient even knowing of the existence of a periproctitis or proctitic suppuration. In other cases, however, pain during defecation, uncomfortable sensations in the rectal region, and a muco-purulent or bloody discharge indicate the character and seat of the lesion.

The diagnosis ought present no difficulty. In every case of periproctitis digital examination of the rectum should be made. In this manner we may not only discover the cause of the disease—e. g., foreign bodies—but its course may be favourably influenced. When the sensitiveness is very marked we should always make a digital examination in the manner previously described. Narcosis is generally unnecessary. If in doubt, we may wait till the time of operation, when the whole field can be carefully examined. We have already pointed out the importance of the bimanual examination (best in the knee-chest position) for eliciting fluctuation.

The diagnosis of *chronic* periproctitis is attended by much less pain for the patient than the acute. A speculum may be employed.

As in proctitis, so here also it may be difficult to establish the cause of the affection (ulcers, fistulæ). In the section on Rectal Ulcers we shall discuss the symptoms of diagnostic importance.

TREATMENT

The treatment is practically surgical. The well-known antiphlogistic remedies (ice and cold applications to the anal region, leeches) are to be applied only in the beginning of the affection—i. e., until the first signs of fluctuation appear. Pain is controlled by injections of morphin, better by opium suppositories (each containing 0.03 gram of the extract, introduced every two to three hours), or by opiates given internally (tinct. opii, 10 to 15 drops t. i. d.).

When fluctuation is present the abscess should be opened as soon as possible. The technic of this procedure is to be found in works on surgery.

3. Rectal Fistulæ

Where an inflammatory process of the rectal mucous membrane ruptures externally and produces a pervious canal, we speak of it as a rectal fistula. This is the so-called complete fistula. Where the canal does not extend through to the skin, we have an incomplete internal fistula. Where periproctitic and ischio-rectal abscesses perforate externally, we have an incomplete external fistula. The accompanying illustrations (Figs. 42, 43, and 44), taken from Esmarch's excellent work[3], illustrate these conditions.

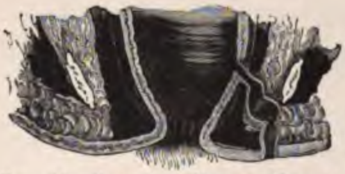

FIG. 42.—COMPLETE RECTAL FISTULA. (von Esmarch.)

The causes of fistulæ are similar to those of proctitis and periproctitis. Ulcerations or abscesses slowly extend through, and finally perforate the different layers of the rectum. The process may develop very acutely, and present the characteristic features

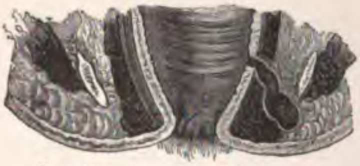

FIG. 43.—INCOMPLETE INTERNAL RECTAL FISTULA.

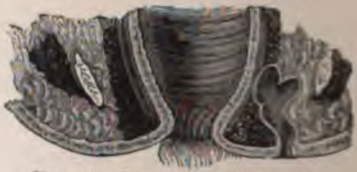

FIG. 44.—INCOMPLETE EXTERNAL RECTAL FISTULA.

of purulent proctitis already described, or it may be very slow and, as mentioned, proceed without the patient's knowledge. According to Allingham's extensive statistics, a large percentage of fistulæ (fourteen per cent) are tubercular. It is very important for the diagnosis whether the fistula occurs in persons who are otherwise well and free from hereditary taint, or whether the fistula is a complication of a general, especially pulmonary, tuberculosis. For the differential diagnosis and for purposes of treatment a careful general examination is required.

SYMPTOMATOLOGY AND DIAGNOSIS

If symptoms of an acute proctitis or periproctitis have been present, we should search for rectal fistulæ. The diagnosis is more difficult when the fistula develops without any previous symptoms. It very frequently happens that patients are only accidentally prompted to have the rectum examined after the disease has lasted a long while. Some discomfort, or a feeling of slight fulness in the rectal region before or after defecation, is generally all that is complained of. When the fistula is a blind one, and the secretions cannot escape, these symptoms may be somewhat increased in severity. There is a sensation of heat in and around the rectum; the patient has a feeling of painful tension, or even of tenesmus.

The *characteristic objective symptom* which disquiets the patient is the discharge of greater or less quantities of pus from the external or internal fistulous orifice. The pus is generally thin and serous, and is rarely mixed with blood or fæces. The discharge may cause an annoying intertrigo. Patients occasionally declare that they pass gas through the fistula. They are weakened by the continual suppuration, feel tired, dispirited, and unwilling to work.

In simple cases the *diagnosis* is easy; in complicated cases it may be so difficult that the kind and extent of the fistulous tract can be recognised only when the patient is under general anæsthesia. A careful examination of the anal region and rectum should be made. The external orifice of the fistula may be variously situated. It is generally in the direct vicinity of the anus, but may be at quite some distance therefrom—e. g., at the perinæum or the gluteal prominence. We occasionally meet with several fistulæ; in fact, the whole region of the anus may be perforated by numerous orifices. The blind external orifice may present a pouting mouth filled with granulations, or it may be a small, barely visible furrow hidden between the anal folds, and scarcely admitting a small probe. The internal opening is recognised by its small indurated prominence, or there may be only a feeling of diffuse infiltration, or no appreciable change in the mucous membrane. In rare cases there is an ulceration at the internal orifice of the fistulous tract. *Complete* fistulæ are recognised by passing probes through the fistulous canal, thus disclosing both its orifices. The best probe for this purpose is the flexible one of zinc recommended by Esmarch; this is gently pushed forward, without the use of any force. Quenu and Hartmann[4] recommend thin, soft bougies, like those used in urethral catheterization. The left forefinger is introduced into the rectum, so that it may follow the direction of the instrument, and thus discover a possible internal opening.

If this does not succeed there may still be a complete fistula, for the probe may have followed a false passage. In such cases a speculum is introduced and a good light thrown into the rectum, and milk or a carmine or eosin solution is injected into the external orifice of the fistula. In this connection it is of practical importance to know that the internal orifice is usually either in the region of the sphincter or directly above it, and that only very rarely is it higher than 5 centimetres above the anal orifice (von Esmarch). In this manner, we may, by careful and perhaps repeated examinations, recognise a complete fistula. This also applies to the incomplete external fistulæ. The diagnosis of an incomplete internal fistula is sometimes much more difficult. The finger when introduced may encounter a small indurated, buttonlike prominence, which can, however, quite easily escape palpation. At all events, where other symptoms indicate fistula, we should not rest until a satisfactory explanation for the symptoms has been found. In complicated cases it will be necessary to make a careful examination under narcosis. The diagnosis of rectal fistula alone is not sufficient. As already mentioned, we must determine its nature. We should search particularly for luetic and tubercular symptoms, which may change both prognosis and therapy.

TREATMENT

The treatment of rectal fistulæ is now purely surgical. In preantiseptic times, when operative treatment of these conditions was a dangerous proceeding, conservative treatment was attempted. The oldest method, known even to Hippocrates and occasionally used to this day, is ligation of the complete or artificially completed fistula. The other methods—scarification, injections of iodin, nitrate of silver, alum, etc.—are all obsolete. With modern asepsis and antisepsis, the operation for fistula has lost all danger. The advisability of operation is doubtful only when there is advanced tuberculosis, and when other tuberculous ulcerations exist in the rectum. In these cases, despite splitting of the fistulous tract, the process steadily progresses. The same caution also applies to carcinomatous fistulæ. In both these latter instances we shall have to limit ourselves to symptomatic treatment, particularly to copious irrigation of the diseased rectum. A description of the technic of the operation for fistula in ano does not belong to this work.

4. Fissures and Spasm of the Anus

Small tears or excoriations at the border of the anus may produce painful reflex spasms of the anal muscles. In the chapter on Intestinal Neuroses we shall see that spastic conditions of the anus may also be caused by hysteria, neurasthenia, or diseases of the central nervous system. *Fissures* are more or less extensive, generally oval losses of substance, which are usually superficial, but which may also affect the deeper muscular tissues. They are often found at the posterior commissure, and less frequently laterally, anteriorly, or in the interior of the rectum. After having lasted a long time, they increase in size, become indurated, and have a dirty gray base. Fissures are generally idiopathic, but may occasionally be produced by gonorrhœa, syphilis, tuberculosis, hemorrhoids, etc. They occur in both sexes, more often in women than in men, and are not infrequently found in early childhood.

Habitual constipation predisposes to fissures, particularly when there is a disproportion between the calibre of the fæces and the anal opening. A soft, irritable skin also favours their development. These conditions are more often present in women, hence the greater frequency of fissures among the latter. According to von Esmarch,* fissures frequently occur in women who suffer from anteversion or retroversion of the uterus. The act of parturition, during which the rectal region and anus are enormously stretched, may easily produce fissures.

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* Loc. cit., p. 148.

SYMPTOMATOLOGY AND DIAGNOSIS

Anal spasm is recognised by the paroxysmal pains which accompany defecation. The patients liken them to that of a red-hot iron boring through the anus. The pain may be limited to the anus, or radiate to the bladder, the external genitals, or the legs. The patients try to suppress defecation and the passing of flatus, so that these cases sometimes have marked abdominal distention. Rest relieves the pain; motion, or even sitting for a long time, may increase it.

The fissure can be discovered by careful inspection or digital exploration of the rectum. Where digital examination is very painful, or where the finger can with difficulty be passed through the anal orifice, it is better to previously relieve sensitiveness by the introduction of a thin suppository of opium and cocain, or by cocain ointment spread over the rectal mucous membrane with the previously mentioned collapsable tin tube. According to von Esmarch, a small polypoid tumour or an edematous fold of the skin of the anus is often found at the external extremity of the fissure. The fissure becomes visible only when this growth or fold is drawn aside. I can recall but one such instance. For the purpose of more thorough examination, surgeons (von Esmarch and others) advise narcosis. Personally I have always found the above-mentioned methods sufficient. It appears to me that narcosis is only indicated in those rare forms in which the fissure is situated higher up. It is of diagnostic importance to determine whether the fissure is idiopathic, or secondary to gonorrhœa, lues, tuberculosis, or hemorrhoids.

TREATMENT

Prophylaxis is of the first importance. In those suffering from habitual constipation, that condition must be treated upon the principles already enunciated (see chapter on Constipation). The tissues of the anal and rectal regions must be hardened by washing with solutions of tannin and alum, or with soaps containing these substances, and by sitz baths. The patients must avoid severe straining during defecation.

When a fissure has been discovered, the anal region must as far as possible be immobilized; the patient should remain in bed till cured. A second preliminary condition to recovery is the artificial prevention of defecation (see page 157). The patients are put upon a fluid diet, and get ten drops of tincture of opium three times daily. Of late I have returned to the use of opium suppositories. If the fissure can be seen, it is best dusted with some dry powder (e. g., airol, xeroform, iodoform, orthoform, calomel, etc.), without directly touching the fissure with the fingers. I have seen more harm than good from washing with antiseptic solutions.

After a week of this treatment I give the patients a large dose of castor oil, and advise them to attempt to pass stool only when they feel that the fæces have become thoroughly softened. Under all circumstances straining must be avoided. The first evacuation is usually painless. Sometimes the entire treatment must be repeated a second or even a third time. I can warmly recommend this method, although some individuals cannot retain their stools for a week; in these treatment is generally unsuccessful. Of 12 cases of fissure whose histories I possess, 6 recovered within eight to ten days, 2 were cured in three weeks, 2 in four weeks, and only 2 had to be operated. I believe it incorrect to regulate the bowels by purgatives.

Local treatment with various astringent, cauterizing, and anæsthetic agents has been recommended. The most frequently used is cauterization with the pure nitrate of silver, or with a ten-per-cent solution of the same. Allingham[5] employs the following:

Calomel	0.25
Pulv. opii	0.10
Ext. bellad	0.10
Ung. sambuci	5.00

♏

This ointment is to be frequently spread over the entire anal surface. Van der Willigen[6], and recently Conitzer[7], recommend pencilling twice daily with a brush dipped in pure ichthyol. I have twice seen good results from this method, but in a third advanced case of fissure it was useless. Cases which are not cured by any of the above methods require radical treatment. A bloodless method consists in stretching and massaging the anal sphincter under narcosis. This procedure, which is often employed in France, and is also highly recommended by Allingham[5] and von Esmarch[8], is rarely used in Germany. The most certain method is the splitting of the fissure with a knife or thermocautery [Paquelin], after which healing is usually quite rapid.

5. Ulcers of the Rectum

Ulcers of the rectum occur under the most varied conditions—primary, secondary, limited to the rectum, or involving other segments of the large intestine.

Primary rectal ulcers are traumatic (from enemata or foreign bodies), gonorrhœal, and syphilitic (both from sodomy). Gonorrhœal and syphilitic ulcerations may also develop indirectly (Bartholinitis, recto-vaginal fistula, breaking down of large condylomata, etc.). Among secondary ulcerations are the tubercular, which always result from auto-infection. Finally, there are the dysenteric and follicular ulcers, which also occur higher up in the intestines. These various types require a brief individual description.

1. *Dysenteric ulcers* result from chronic dysentery, ulcerative destruction of the rectal mucous membrane, or from a follicular catarrh. By destruction of the follicles and confluence of the destroyed areas there gradually develop deep and extensive ulcerations. The rectal lesion is always secondary to involvement of other portions of the large intestines. The ulcers may extend into the serous coat, and produce perforative peritonitis, abscess, and fistula, or subsequent cicatricial stenosis.

2. *Follicular ulcers* develop in the rectum and large bowel where there is catarrh with marked swelling of the follicles. When these follicles rupture, a small flat ulcer results. Several such ulcers coalesce and produce larger ulcerations, which, by the persistence of the catarrh or through other unfavourable circumstances, may become quite deep, and even rupture into neighbouring organs. As von Esmarch[3] states, these ulcers are sluggish in character, heal badly, and lead to the formation of cicatrices and polypi. According to this authority, most cases occur toward the end of exhausting diseases or after severe injuries and operations, and produce death by colliquative diarrhœas.

Follicular ulcers are frequent in children with chronic diarrhœa. I have never observed this form of ulceration in adults, not even in those suffering from severe catarrh of the large intestine. On the other hand, I have repeatedly seen chronic follicular swelling, with pain and other disturbances, in catarrh of the large intestine and rectum.

3. *Tubercular ulcers* (see Fig. 45). These result from swallowed tubercular sputum, from tubercle bacilli which have reached the rectum by means of the blood and lymph channels, or from

mechanical insults to the anus or rectum. In the case from which the accompanying illustration is taken, the probable cause of the lesion was continued contact of the anal mucous membrane with mechanically and chemically irritating evacuations. The patient suffered from intestinal tuberculosis. The ulcers are formed by the breaking down of tubercular nodules (lenticular ulcer). Similar to tubercular ulcers of the rest of the intestine, those of the rectum are characterized by a circular arrangement. Near the ulcers fresh tubercular, grayish, globular nodules occasionally develop, undergo the same necrotic process (softening, fatty and caseous degeneration), and produce secondary tubercular ulcers. We have already mentioned that tubercular ulcerations tend to spread, and to cause periproctitis and rectal fistulæ.

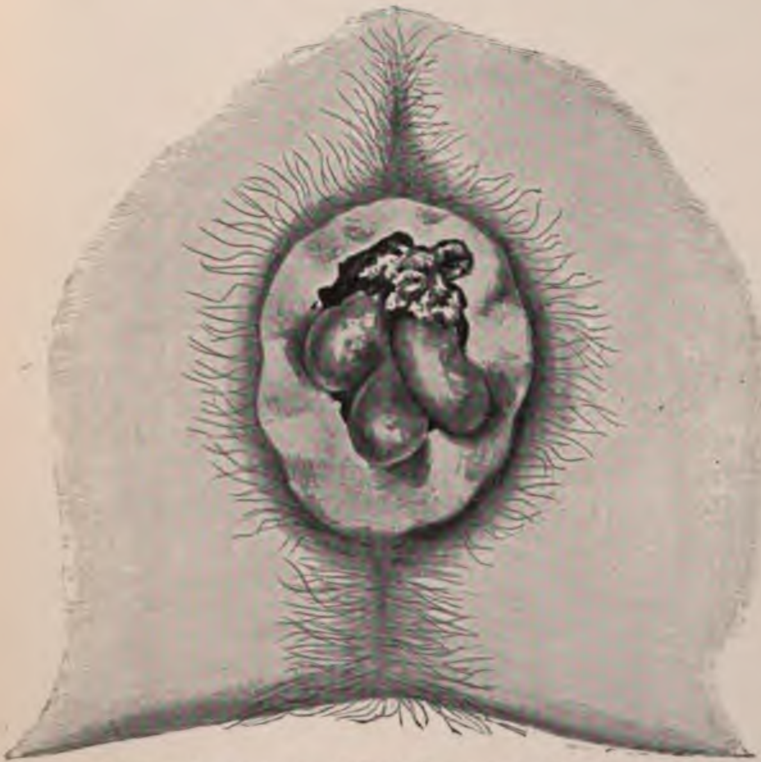

FIG. 45.—TUBERCULAR ANAL AND RECTAL ULCER, WITH HEMORRHOIDAL NODULE. (Original observation.)

4. *Syphilitic ulcers* of the rectum occur in the most varied

forms. They are found as soft chancres (primary, from coitus per anum; secondary, from infection by chancroidal secretion) or as hard chancres, either as a primary infection (very rare), or more frequently by the breaking down of broad condylomata in the neighbourhood of and spreading toward the anus. They may occur as gummatous ulcers. Gummata may be found in various stages: true gumma, degenerated gumma, superficial ulcer, or scar tissue from old healed ulcerations. Frequently we can also distinguish the more recent processes (rounded nodules with beginning ulcerative degeneration, and containing necrotic, brownish-red masses) from the older, deep, irregular ulcerations.

In constitutional syphilis there may be a proliferation of the deep layers of the connective tissue, a condition von Esmarch[3] designates as gummatous or syphilitic polypi. Unlike true rectal polypi, these do not originate in the mucous membrane, but from a proliferation of the submucous, submuscular, or subserous cellular tissue. Virchow has therefore called them granulation tumours (granulomata). When these heal, cicatrices, which may produce extensive stenosis, develop; these will be described later. In marked cases the entire rectum is changed into a rigid, immovable funnel with immensely thickened walls. If the lowermost portion of the rectum is affected, complete destruction of the sphincter may result.

5. *Gonorrhœal ulcers.* Opinion is divided as to whether rectal ulcers may be gonorrhœal or not. While Jullien[1] believes in their existence, Baer[2] considers them complications (post-gonorrhœal ulcerations), particularly since no gonococci have been demonstrated in the excised portions. According to Baer, these ulcers are usually situated upon the anterior or posterior wall of the anal orifice upon a protrusion of its mucous membrane or of a hemorrhoidal fold. The surface of the ulcer is always directed toward the lumen of the rectum.

SYMPTOMATOLOGY AND DIAGNOSIS

The first symptom observed in uncomplicated ulcer of the rectum is a change in the evacuations. The stools are generally thin and fluid, are mixed with blood and pus, have a very fetid odour, and may consist only of blood and pus. In addition, there is more or less marked tenesmus and pain radiating to the bladder, back, legs, and genitals. The subjective symptoms may be absent, or may not be prominent. As a result of hemorrhage and suppuration, and occasionally from fever, the patients lose considerable flesh and strength. From the symptoms above enumerated we may assume the presence of ulcerative processes in the rectum. For positive diagnosis a local examination is, however, necessary.

Careful digital exploration, whereby the situation and nature of the ulcers can be fairly well determined, is the best method of examination. It is also desirable to inspect the mucous membrane by means of a speculum, the rectum being meanwhile irrigated with water. In this manner the diagnosis of tubercular, syphilitic, or catarrhal ulceration can usually be made although great difficulties may be encountered.

A thorough history and careful study of the other clinical symptoms are of the utmost importance, particularly in the differentiation between syphilitic, dysenteric, and tubercular ulcers. We must always search for other signs of syphilitic or tuberculous disease. When ulcers appear gonorrhœal, the secretion should be examined for gonococci. Similarly the tubercle bacillus must be looked for. Small portions of the ulcers may have to be excised for microscopical examination, although positive results are not always obtained. Since ulcerations may also occur from ruptured abscesses (salpingitis, Bartholinitis), vaginal examination must never be omitted.

TREATMENT

The treatment of ulcers of the rectum is very tedious. The reason is evident. The rectum offers the best chances for the propagation of ulcerative processes, but the worst for their cure. The long list of remedies that have been recommended bears out the above assertion.

A priori, the aim of our therapy would seem simple enough—viz., wherever possible, to eradicate the underlying cause. Where this is not possible, energetic local treatment should be instituted.

Prophylactic treatment is often employed in tuberculosis and syphilis, but, unfortunately, with little success. The chief cause for this is that patients regard the first phases of their rectal affection too lightly, and therefore present themselves for treatment when the disease is already far advanced. In tubercular rectal ulceration the patients usually have pulmonary or intestinal tuberculosis, so that treatment will be of little avail.

The conditions are more favourable in ulcerating chancres; these may be healed by appropriate anti-syphilitic treatment (Köbner). Although occasionally cured by energetic use of iodids by mouth and rectum, gummatous ulcers do not as a rule respond well to treatment.

The treatment of gonorrhœal ulcers is the same as that of anal gonorrhœa—i. e., astringent and antiseptic solutions. According to Baer and others, the results are not more satisfactory than in chronic urethritis.

Symptomatic treatment should be directed toward the increase of general health and strength and the regulation of the bowels. The most difficult therapeutic problem is the cure of the ulcers by local treatment. The number of preparations recommended for this purpose is so large that we can only mention the most important and usual ones. These include nitrate of silver, sulphate of zinc, tannin, aceto-tartrate of aluminium (Boas), zinc-chlorid solutions, and carbolic acid. They are best applied in the form of rectal irrigations. Allingham recommends the use of soft ointments applied by a specially constructed ointment spray.

The difficulty in local treatment lies in the fact that in advanced cases constrictions and dilatations almost always prevent remedial agents from reaching the main seat of the disease. Surgical methods have therefore been attempted. In marked stricture of the anal orifice, in order to render the diseased area more accessible, posterior sphincterotomy has been proposed. In ulcerations higher up, the most appropriate treatment would seem temporary colostomy, with subsequent local treatment through the intestinal fistula. Except in cases of stenoses (see below), but little experience with this operation has been gathered. When, as in rectal stenosis, the disease is very extensive and obstinate, and remains uninfluenced by all palliative measures, the only remaining procedure is resection of the ulcerated portion.

6. Strictures of the Rectum

The causes may be external or internal. External conditions are disease of neighbouring organs, neoplasms, plastic exudates, and vesical calculi. Internal stricture may be produced by simple obstruction of the lumen of the rectum, by fæcal masses, foreign bodies, enteroliths, prostatic hypertrophy, and tumours, or by inflammatory conditions of the rectum itself. The latter are undoubtedly the most frequent causes of stricture, and therefore merit thorough discussion.

All lesions of the rectum which heal by granulation must produce narrowing of its lumen. Wherever a stricture has developed, its increase is favoured by continued irritation of the adjacent tissues or by an extension of the underlying disease.

We have previously described (page 494) the various kinds of ulcerations of the rectum. Syphilitic and dysenteric ulcers are far more apt to produce extensive stenosis than are tubercular. To what extent gonorrhœal processes produce stenosis is still a disputed question.

No doubt there is an inflammatory rectal stenosis similar to inflammatory pyloric hypertrophy or to the so-called multiple submucous sclerosis of the French. In his latest work, Bushe * describes a very characteristic case. Rieder[3] also believes in the existence of a chronic inflammatory proctitis with destruction of the mucosa and proliferation of the submucosa and muscularis.

The stricture is usually situated directly above the anal orifice, but may even be as high up as the sigmoid flexure. (Several years ago I saw an instance of this latter condition in a young woman. The etiology of the case could not be determined.) The strictured segment may be either straight, corkscrew shaped, or arborescent. The wall of the canal may consist of thick, indurated mucous membrane without ulcers, or there may be numerous primary or secondary erosions. In the most complicated cases all possible pathological changes may be combined—viz., proctitis, periproctitis, fissures, hemorrhoids, fistulæ, etc.

The greatest number of rectal stenoses are found in women with present or previous syphilitic symptoms. In the former instance syphilitic ulcers or gummata may be found on the genitals and in the rectum, or in the rectum alone. These conditions are not difficult to diagnosticate, although the manner in which the syphilitic virus infects new areas may not be apparent. According to Quenu and Hartmann[4], the lower group of rectal veins anastomose directly with branches of the external pudendal, which latter originate in the posterior commissure of the vulva, the chief seat of syphilitic infection.

The question of post-syphilitic strictures has caused very much discussion, and at the present time all etiological factors have not been agreed upon. We can, however, safely say that syphilis is undoubtedly the cause of a large number of rectal strictures. The fact that the above lesions frequently occur with other syphilitic stigmata (exostoses, amylosis, endarteritis, syphiloma of the liver,

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* Loc. cit., page 109.

etc.) indicates their close association with constitutional syphilis. The objection of the opponents of this theory (Nickel[9], Polchen[10]), that rectal stenoses are refractory to specific treatment, is hardly convincing. The theory that rectal ulcers are produced by traumatism does not explain their greater occurrence in women. We admit that, besides syphilis, gonorrhœa and catarrhal inflammations of the rectal mucous membrane may produce strictures. Rieder[8] has recently demonstrated that there is a proliferation of the intima of the rectal veins, even to complete obliteration, while the arteries remain intact. This condition he considers characteristic of syphilitic rectal stenosis.

SYMPTOMATOLOGY AND DIAGNOSIS

Obstruction to defecation is the foremost symptom of stricture of the rectum.

As in carcinomatous strictures (already discussed on page 318), the early symptoms of these rectal strictures are obscure and generally escape observation. The patients first present themselves for treatment when symptoms of ulceration or of increasing rectal stenosis become prominent. In the former instance there is a bloody, mucous, or purulent discharge; in the latter, painful tenesmus with fragmentary, scybalous, small-calibred or semifluid dejections. The symptoms of ulceration may be accompanied by those of stricture. Above the stricture (in the sigmoid flexure or higher) abdominal palpation reveals fæcal masses, which soon produce a sacculated dilatation of this part of the large intestine—an important symptom of low strictures. There may be so-called "false diarrhœas," which are easily mistaken for intestinal catarrh.

The *general health* is gradually undermined by the tenesmus and loss of blood and pus. It is surprising, however, to note how long the general condition of patients with non-malignant strictures remains good. This applies also to malignant strictures, though here we may generally detect the characteristic cachexia. Acute, complete stenoses or perforations into the neighbouring organs may take place, but are rare. The lower the stenosis the more infrequent these perforations.

Continued ulceration may cause extensive destruction of the sphinctèr ani. The symptoms of rectal incontinence then appear, tenesmus ceases, and there is a profuse, continuous discharge of blood, pus, and mucus.

The *diagnosis* of rectal stricture is readily made. Upon introduction, the finger immediately strikes an obstacle; at the same time we are informed of the degree of the stenosis, at least of its lower (anal) portion. To determine the size of the stricture and the condition beyond, the introduction of rectal bougies is indispensable. As already mentioned (page 81), we may use various instruments for this purpose. We should always remember that we are introducing the instrument into a cavity whose length, direction, and lateral recesses are unknown, and that by this procedure we cannot determine the condition of the rectal wall and the presence, extent, and depth of the ulcerations. Considering all these facts, we may ask whether the diagnostic value of instrumentation is as great as its dangers. It is plain, however, that therapeutic bougieing has to reckon with other factors. Bougieing is only absolutely necessary in strictures beyond the reach of the examining finger, for here the diagnostic difficulties cannot otherwise be overcome. Thus the previously mentioned case of sigmoid stricture (page 494) had passed through many hands before the correct diagnosis was made. We have already pointed out the characteristics of stricture of the sigmoid flexure and of the upper part of the rectum (page 82). I agree with experienced clinicians that the use of a speculum is generally unnecessary for the diagnosis of low-seated strictures, although one may thus obtain a better idea of their character.

The examination of the dejections, their form, and more especially the admixture of purulent, fetid, bloody masses, completes the clinical picture and diagnosis. As already stated, changes in the stools are often the first symptoms to alarm the patient, and should always lead to careful local examination.

Differential Diagnosis

The etiology of strictures is much more difficult to determine than is their diagnosis. Since they will scarcely escape a careful examination, we need not here consider external tumours, exudates, etc., which constrict the rectum. Prostatic tumours and enlargements will likewise rarely cause error in diagnosis, but differentiation between malignant and benign rectal strictures, particularly between carcinomatous, luetic, gonorrhœal, and dysenteric varieties, is quite difficult.

In distinguishing between malignant and non-malignant stenoses, the history and clinical course of the disease are frequently of value. The existence of syphilitic infection or of a previous dysentery can usually be established. The course of the affection is more important. The patients may state that their symptoms date back several years—a fact of great significance as regards the kind and character of the stricture. The age of the patient and the absence of cachexia may to some extent speak for one or the other type of stenosis.

As we have seen in the section on Cancer of the Rectum (page 321), digital examination may give us useful information—e. g., in the differentiation between carcinomatous and syphilitic strictures. Nevertheless, errors may occur. In such cases excision and microscopical examination of a rather large piece of the new growth may make the diagnosis positive.

The differential diagnosis between syphilitic and dysenteric rectal strictures is very difficult. In his work on tumours, Virchow[2] states: "Gummatous ulcers resemble diphtheritic, and more especially dysenteric, ulcers. This similarity is so marked that I have often been in doubt as to whether the destructive process in a given case was syphilitic or dysenteric. The same is also true of strictures. The site of the lesion may to a certain extent guide us. In dysenteric processes the lesions are more frequently found in the sigmoid flexure; in syphilitic, in the ampulla of the rectum or close to the anus. In addition, the more even and broader ulcerations of syphilis contrast with the eroded, irregular, superficial and deep ulcerations of dysentery." In view of the rare occurrence of dysentery in our country [Germany] the clinical differentiation will usually be easy.

Tubercular rectal stenoses are rare, and are generally accompanied by signs of tuberculosis in other portions of the body (lungs, peritoneum, other intestinal segments, genito-urinary system, etc.); the etiology of the ulcers may be determined by examination of the secretion for tubercle bacilli. When suspecting gonorrhœal stricture, gonococci should be sought for. It must not be forgotten, however, that, especially among prostitutes, the simultaneous occurrence of syphilis and gonorrhœa is by no means rare.

From the above data we may (perhaps after a long-continued observation), in many cases, make a probable or even a positive diagnosis.

TREATMENT

Whatever the nature of the underlying process, internal treatment of strictures of the rectum is useless. Most experienced clinicians agree upon the futility or slight value of antisyphilitic treatment. According to dermatologists, in order to prevent cicatricial contraction, radical antisyphilitic treatment is always indicated in fresh gummatous syphilis. In the majority of cases we must alleviate stenotic symptoms by laxatives. Drastic drugs are to be avoided. Rhubarb, frangula, flowers of sulphur, compound licorice powder, or magnesia usta, in conjunction with the so-called "constipation diet" (see page 146), are usually sufficient. If, as determined by external palpation, there is long-standing coprostasis, the safest procedure is the administration of large doses of castor oil (2 to 3 tablespoonfuls, repeated, if necessary, for several days).

The best palliative measure is methodical dilatation of the stricture by rectal bougies. Views differ widely regarding the value of this method. Most surgeons do not adopt the extreme view of Schuchardt, Rieder, and others, that bougieing should be entirely discarded. In several cases of very advanced luetic stricture I have achieved remarkably favourable results with sounds, although the treatment had to be carried out three times a week for several months. I consider proctitis or periproctitis positive contraindications to the use of bougies. In a dissertation upon the clinical material of G. Lewin, Alderhot[12] also speaks in favour of bougieing. Before beginning treatment, it is advisable to point out to the patient the length of time required, as well as the possibility of relapses after bougies have been discontinued. If the patient is intelligent, he may after a few weeks be intrusted with the instrumentation himself, and the result occasionally controlled by the physician. We begin with the smallest bougie that will pass the stenosis, gradually increasing the size. The instrument should be left *in situ* several minutes, and, to aid in the dilatation, should be given gentle rotary movements.

The best bougies are solid soft-rubber ones, purchasable everywhere, or Hahn's hollow bougies containing a spiral frame. The latter are not soft enough to give way when they encounter the obstruction, but are sufficiently elastic not to cause laceration. If properly curved (Bushe), the following instruments are also recommended: French bougies, hard-rubber or glass bougies (von Esmarch), and olive bougies fashioned after Trousseau's sounds. Credé and Körte prefer curved, hard-rubber bougies. Because of their lack of danger soft instruments are to be preferred. Where discharges of blood or pus weaken the patient, we may try astringent irrigations (aceto-tartrate of aluminium, tannin, nitrate of silver); for obvious reasons, however, such irrigations are of little benefit.

If the above palliative measures cannot be applied, or have proved futile, we should advise operative treatment. There are several methods; opinion regarding their respective value is as yet divided.

These methods are the following: (*a*) Lateral incision with a scalpel in order to render bougieing easier. This has not met with great favour, since superficial incisions are of little use and deep ones dangerous. (*b*) In annular cicatricial stenosis of the anus, Dieffenbach advocated extirpation of the stricture and drawing down and suture of the distal mucous membrane to the lower edge of the wound. (*c*) In very marked stenosis of the anal region and of the lower portion of the rectum, Péan recommended that the canal should be cut longitudinally and the cut edges sutured transversely, similar to the pyloroplasty of Heineke-Mikulicz. This method, however, presupposes a stenosis of equal degree throughout and the absence of fistulæ, and hence will be of use in only a limited number of cases. (*d*) Colostomy. This is usually not dangerous, but is functionally unsatisfactory. The method first used by Thiem[13]—temporary colostomy and subsequent bougieing of the stenosis—gives much better results. As soon as the stenosis is cured (cure naturally proceeding much more quickly under these circumstances) the artificial anus may be closed. At present this is the best and undoubtedly the most preferable procedure, because it is not dangerous, and gives the best functional results. It is doubtful, however, whether the majority of stenoses can be sufficiently dilated to permit the normal passage of fæces within a reasonable time. Further experience in this field is necessary.

Sonnenburg[14], and recently Rotter[15], have proposed two methods whose value can only be determined by future operations.

(*e*) In very extensive syphilitic (and gonorrhœal) strictures, which, owing to their high situation and extent, cannot be extirpated, Sonnenburg recommends extirpation of enough of the coccyx and sacrum to lay bare the callous stricture and its surroundings. On account of the extensive adhesions present, the peritoneum is not endangered. The whole length of the stricture is then divided from without inward; the sphincter is not divided. The wound is tamponed, and heals slowly. Later, long-continued bougieing is necessary. Sonnenburg calls this operation "*external rectotomy.*"

(*f*) Following the principle recommended by the American surgeon Bacon, Rotter connects the intestinal segment above the stricture (the sigmoid flexure portion) with the normal segment lying between the stricture and sphincter ani. In two out of three cases this operation was successful, and was accompanied by good functional results. Rotter calls his operation "*sigmoid rectotomy*." Aside from the fact that this procedure circumvents but does not remove the stricture, it has a limited application, for it can only be used when there is enough healthy rectum above the sphincter ani for implantation of the sigmoid flexure and it is no longer possible to treat the stricture.

(*g*) The most thorough and at the same time most severe operation is *resection of the rectum*, first performed by James Israel in 1885, and since then repeatedly by Schede[16] and many others. When we consider the difficulties of this operation, the results achieved by Schede are quite satisfactory. In 17 cases from Schede's clinic, recently reported by Rieder[8], none died from the operation. Permanent results were obtained in 10 cases; of these, 5 remained cured and free from recurrence; the remaining 5 had either relapses or fistulæ. Of the 5 cured cases (4 of them were positively syphilitic) 1 was well since one year, 2 others since two years, and 2 since six years. It is very difficult to obtain a good functional result after the operation.

It is only from rectal examination that we can, in a given case, determine which of the above operations is the most appropriate. Besides, the surgeon is swayed by his preference for one or the other operation, and by his own results.

7. Prolapse of the Rectum (*Prolapsus Recti*)

The rectum is so closely connected with surrounding tissues that only under special conditions is this attachment loosened. Such conditions are for the most part found in children. According to the statistics of Bokai[17], of 350 cases of prolapsus recti, the greatest number occurred in children in the second and third years of life, the first and the later years showing a markedly diminished predisposition to the affection. Other prominent etiological factors are poor general health, constipation, respiratory diseases (especially pertussis), and catarrh of the large intestine (particularly of the rectum) with severe tenesmus.

In adults, besides the above factors, there are dysuria, unnatural coitus, senile atrophy of the muscles of the pelvic outlet (levator ani, rectal sphincters, and retractors); in women, overdistention of the muscles of the pelvic floor, etc.

The prolapse generally develops gradually; at first the anal mucous membrane protrudes (as it does physiologically in the horse during defecation); later there is a protrusion of all the layers of the rectal wall. In extensive rectal prolapse the peritoneal fold of Douglas is also drawn down. The sac formed in this manner may, in rare instances, contain intestine, ovaries or bladder—a condition known as rectal hernia. True rectal is to be sharply distinguished from hemorrhoidal prolapse, the latter being easily differentiated by the well-marked bluish nodules. We shall later return to the subject of hemorrhoidal prolapse.

Symptomatology and Diagnosis

The first symptoms are usually not well marked, and hence have rarely been observed. The patient's attention is directed to the lesion only when large portions of the rectum prolapse and are not spontaneously reduced. On straining, we may then distinctly see the rosettelike protrusion of the mucous membrane with the central opening from which fæces are emptied. When the prolapse is reduced the examining finger can easily recognise the relaxation of the sphincters. At first the prolapse occurs infrequently, and only from severe straining at stool; gradually the resistance of the sphincters is more easily overcome, so that coughing, laughing, sneezing, and even walking, cause protrusion.

In consequence of mechanical irritation, particularly in the beginning of the disease when the rectum is unaccustomed to any foreign influences, inflammation with subsequent catarrh of the mucous membrane develop. A copious discharge of viscid mucoid secretion follows. Accompanying this condition there may be hemorrhages, ulceration, and, where long-continued incarceration exists, gangrenous inflammation with necrosis of the prolapsed segment and serious sequelæ. In this manner spontaneous cure often results. In the last stages of the disease, having gained a certain amount of practice in the rapid reposition of the prolapse, the patient becomes more or less accustomed to the condition.

We can gauge the age of the prolapse by its characteristics. During the first stages it is succulent, soft, covered with mucus, and rich in blood supply; later it becomes tough, smooth, and resembles epidermis. This latter condition favours easy reduction.

The occurrence of prolapse with simultaneous descent of abdominal contents is of clinical importance. In such cases the prolapse increases in size; the orifice of the rectum is then pushed toward the coccyx, and does not regain its axial position till the hernial contents are reduced. If, as the result of marked distention or inflammation of the rectum, or of abnormal tension of the sphincter or levator ani, the intestinal contents become incarcerated, all the sequences of intestinal strangulation may ensue.

The *diagnosis* of prolapsus recti is rarely difficult. Careful examination will nearly always prevent error in differentiating between rectal and hemorrhoidal prolapse. In extreme rectal prolapse it is often difficult to determine whether or not intestinal contents, etc., are present in the peritoneal sac. There are numerous reports of unsuccessful operations due to this error. Careful and repeated examinations (preferably under anæsthesia) are therefore necessary.

Intussusception of the colon must be considered, as it frequently occurs during the earlier periods of life. There is considerable difference in the course of these two diseases (see page 387), and difficulty in their differentiation can only arise when we have neither reliable history or observation to guide us. In intestinal invagination the examining finger can feel the rectum outside the intussusceptum without meeting the point of reduplication. It is important also to note that in prolapse there is a sort of furrow between the base of the prolapsed portion and the anal ring, which furrow disappears only in long-standing cases. In a prolapsed and invaginated colon a bougie may be introduced for a long distance, while in rectal prolapse the instrument's progress is soon stopped.

TREATMENT

This is most successful during the early stages. At such times cure may be obtained by regulation of the bowels, careful avoidance of straining, and by local tonic treatment (cold irrigations with astringent solutions, etc.). Unfortunately we usually see cases for the first time when they are far advanced, and when they are much less amenable to conservative treatment.

The age of the individual and the duration of the prolapse have an important bearing upon the treatment. In very old prolapses, where operation is declined, internal treatment can only be symptomatic. In children in whom the prolapse is not very far advanced, active internal or surgical measures are always indicated.

Besides careful regulation of the bowels (one semisolid movement daily) and thorough regional treatment with astringents, local subcutaneous injections of ergotin (0.1 gm. to 0.2 gm. per dose) or strychnin (0.001 to 0.002 gm.) may be given. This treatment has often been successful, and is worthy of systematic trial. Should it fail, surgical measures must be employed. The rectal support of von Esmarch (see Fig. 46) is the best means of controlling the prolapse in patients who complain mainly of the discomfort.

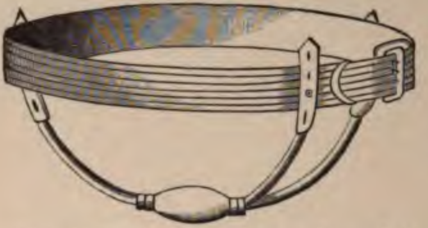
FIG. 46.—RECTAL SUPPORT. (von Esmarch.)

In my opinion, the indication for surgical treatment, which nowadays has entirely superseded the former bloodless methods (actual cautery,* cauterization with mineral acids, etc.), depends upon the severity of the symptoms. The prolapse *per se* is certainly no indication for operation, for I have seen patients become entirely accustomed to their condition.

The question of operation is quite different where there is difficulty in replacing the prolapse, where there is any inflammation or hemorrhage, and where the bowel protrudes in the interval of defecation. We should lose no unnecessary time with palliative treatment, especially since the present state of surgery has robbed this operation of its dangers. Unfortunately, operation does not always guard against relapses. For the various operative procedures the reader is referred to surgical text-books.

8. Hemorrhoids

These are diffuse or circumscribed dilatations of hemorrhoidal veins situated in the subcutaneous tissue of the outer anal region and in the submucous tissue of the lower rectal segment. The old classification of hemorrhoids into external, internal, and mixed groups still holds good. In mixed hemorrhoids the extra-rectal portion is usually small and the intra-rectal portion well developed. We find the statement, particularly in older literature, that hemorrhoids may extend over the entire rectum and even into the sigmoid flexure. If this condition occurs at all, it certainly is extremely rare. For the most part hemorrhoids are undoubtedly caused by hindrance to the return of blood to the vena cava and portal vein. We distinguish,

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* [This certainly is a surgical procedure—in fact, the cautery operation at the present time is the favourite method in the United States for uncomplicated rectal prolapse.—TR.]

1. Internal causes, within the rectal mucous membrane.
2. External causes, which act by compressing the hemorrhoidal plexus.
3. Disturbances of the general circulation.

Internal factors produce three quarters of all hemorrhoidal formations. Fæcal stasis is the most important and frequent of these causes. A vicious circle develops; coprostasis, pressure on the hemorrhoidal veins (further increased by straining of the abdominal muscles), and formation of hemorrhoidal nodules, followed by mechanical intestinal stenosis, stasis, proctitis, increased constipation, etc.

Other changes in the mucous membrane of the rectum and other parts of the large bowel (stenosis, new growths, foreign bodies, prostatic enlargement), by preventing the normal passage of fæces, may give rise to hemorrhoids. As in cancer of the rectum, we may observe the paradox of a diarrhœa due to coprostasis above a stenosed segment. Hemorrhoids are also found in chronic diarrhœa, particularly in catarrh of the large intestine. In such cases the hemorrhoids may be caused by the tenesmus, hyperæmia, and perhaps also by the inflammation of the rectum and lower segments of the large intestine.

The *external* causes of hemorrhoids are tumours of the neighbouring organs which retard the rectal circulation. The simplest example of this is pregnancy, during which, according to Budin[18], 35 per cent of all hemorrhoidal cases develop. These disappear after the puerperium. Tumours of the uterine adnexa, retroflexion and tumours of the uterus, disease of the urethra and bladder, particularly such as affect the contractility of the latter organs, may also produce hemorrhoids.

Formerly *disturbances of circulation* (third cause) were regarded as the chief source of hemorrhoids. Even at present similar statements are found in almost all text-books of special pathology. Nothnagel[19] mentions the rarity of hemorrhoids in stasis of the portal system, and in diseases of the heart and lungs. He states that in cardiac insufficiency the pathological increase of pressure is spread over so large a vascular area that it would scarcely affect the hemorrhoidal plexus in the manner formerly assumed. My own experience also speaks against the theory of hemorrhoids from general vascular congestion. Even if the two conditions coexisted it would still have to be demonstrated that the hemorrhoidal complication was not due to the constipation present.

Bouchard found hemorrhoids in 28 per cent of cases of cholelithiasis. No comment on this is found in the classical monograph of Naunyn, and, as far as I know, this observation has not been confirmed. It does not agree with my own experience. That hemorrhoids may occasionally occur in cholelithiasis is not remarkable, for it is well known that a large proportion of these patients suffer from constipation.

Heredity is often mentioned as a predisposing factor in hemorrhoids, but in all probability there is really hereditary intestinal atony, a condition not at all infrequent.

It has often been demonstrated that a sedentary life predisposes to hemorrhoids, but here again the hemorrhoids result from habitual constipation.

Age and Sex.—That hemorrhoids is really a disease of advanced life follows both from the underlying conditions producing the disease, and from the lessened elasticity of the vessels at this time of life. (I might mention that Lannelongue[20] reports a case of hemorrhoids in a newly born infant.) It is quite striking (and my observations upon numerous cases confirms this) that in the majority of instances hemorrhoids are more frequently found in men, whereas habitual constipation occurs oftenest in women. Besides this, the occupation of women is more restful and quiet than that of men. I can only explain this fact by the numerous venous plexuses in the female genitals, which, while not entirely preventing, certainly make the occurrence of varicose conditions very difficult.

In my opinion, the oft-mentioned distinction between stout and thin anæmic hemorrhoidal patients will as little withstand scientific criticism as the distinction between stout and lean persons with diabetes or constipation. This theory is a remnant of the old teaching, that hemorrhoids exert a beneficial influence upon the general system. It is true, as Nothnagel says, that we find hemorrhoids much more rarely in stout individuals, but this is solely because the tense, firm connective tissue here present greatly retards the development of venous dilatation. Conversely, in cachectic individuals who do not suffer from severe constipation we may often observe hemorrhoids.

Anatomically we differentiate between diffuse dilatation of the rectal veins and true hemorrhoidal nodules. The first generally form a visible rosette, consisting of dilated, often spirally twisted veins, under the skin or mucous membrane. Hemorrhoidal nodules, on the other hand, vary greatly in size, and are single or multiple. When multiple, they surround the anus circularly or wreathlike, and are either sessile or pedunculated. Inflammatory adhesions may produce a confluence of the hemorrhoids, and, as a result, large, almost angiomatous masses are formed. The hemorrhoids may, however, atrophy, and become covered with epidermis. Their original character can then only be recognised by their bluish colour, their consistency, and their characteristic arrangement. By thrombosis and calcification of the veins so-called phleboliths may be formed.

At present our views regarding the histological character of these varices differ. We shall not enter into a minute discussion of this subject, but only mention that Reinbach[21], who has recently carefully studied the histological structure of hemorrhoids, concludes that they are not varicose veins, but true benign tumours—i. e., angiomata.

Hemorrhoidal may conduce to rectal catarrh, with more or less marked mucous secretion (so-called "mucous hemorrhoids"). Fissures and excoriations occur quite frequently, and may lead to abscesses, fistulæ, and (rarely) to very severe inflammation and peritonitis.

SYMPTOMATOLOGY

The former view that hemorrhoids was a constitutional disease (even nowadays we speak of a "status hemorrhoidalis"), explains why the description of this disease was unnecessarily extensive. Many indefinite abdominal disturbances and circulatory and respiratory symptoms were attributed to hemorrhoids. Venesection, which prevailed even to the middle of this century, was one of the consequences of these theories.

At present hemorrhoids are viewed from a purely local standpoint; we treat the hemorrhoidal nodule, and not the hemorrhoidal diathesis. We must not entirely relinquish the old theory of a general disturbance brought about by the "status hemorrhoidalis." We must admit that certain definite symptoms closely associated with altered blood pressure are produced by the hemorrhoidal varix; but many mild and (particularly in cachectic persons) severe cases run their course without symptoms. When present, the symptoms are generally limited to the diseased area, although, as already intimated, they may extend to other regions of the body.

The *local* symptoms are constipation, a feeling of pressure and heaviness in the rectum, tenesmus, and itching and burning in the anal region. Defecation usually brings relief. In external hemorrhoids there is also more or less discomfort in sitting, even upon a soft pillow, and in riding, bicycling, jumping, and gymnastics.

These symptoms may be accompanied by *hemorrhages*, slight in amount or (rarely) sufficiently severe to cause the most profound anæmia or even death. The bleeding may recur periodically. Hemorrhages are often preceded by increased hemorrhoidal symptoms—feeling of congestion, tenesmus, severe pains radiating to the bladder, etc. With the onset of the bleeding these symptoms disappear.

Strangulation of protruding hemorrhoidal nodules constitutes one of the most painful complications. The masses which have been protruded from the anal fold by straining usually return easily, either spontaneously or by manipulation. They may, however, remain prolapsed, and become swollen and inflamed; an inflammatory œdema then develops about the anus; the patients suffer unbearable pain, and, as in strangulated hernia, may collapse. If strangulation is not relieved, the nodules may become gangrenous and necrotic, or a purulent inflammation with its serious sequences may result.

In very old cases, in consequence of paresis of the sphincter, the nodules may prolapse in walking, bending, coughing, laughing, sneezing, etc.

As already mentioned, inflammation of intra-rectal hemorrhoids may produce symptoms of acute proctitis—severe pain, repeated and increasing tenesmus, sphincteric spasm, etc., and occasionally fever.

General disturbances are more apt to occur in chronic hemorrhoidal disease. Besides chronic anæmia many patients suffer from abdominal fulness and pressure, necessitating the loosening of clothing. The passing of flatus affords temporary relief. There may also be severe pain in the back, increased by bending or other active motion. Some patients complain of sciatica, and resort unsuccessfully to bath treatment. Certain nervous symptoms are also present—a feeling of fulness in the head, dizziness, nausea, floating bodies before the eyes, etc.

These symptoms may be considered neurasthenic, or, to be more modern, evidences of auto-intoxication, or perhaps as the result of habitual constipation. We must admit that there remain symptoms which can only be explained by changes in blood pressure in the vena cava and portal vein. If we remember that an accumulation of gas in the stomach or intestine not only gives rise to local discomfort, but also to general disturbances—pressure in the head, feelings of fear, palpitation, etc.—and if we recall the disturbances that occur in the beginning of menstruation, we must acknowledge the correctness of this view. The disappearance of these symptoms, with the relief of the constipation is no proof that they depend upon that condition, for the hemorrhoidal affection is at the same time favourably influenced.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

In the great majority of cases the diagnosis is easy. Under no circumstances must it be based entirely upon the statements of the patient. Inspection of the anal region alone is not sufficient; the rectum must always be included, and, in women, the genital organs. Treatment and prognosis are considerably influenced by the results of such examination. If internal hemorrhoids are suspected, they are best brought to view by having the patient strain strongly. It is best to first give an enema of warm salt water, and then to have the patient strain while sitting upon a chamber filled with warm water. If this does not succeed, a rectal speculum must be used. We should avoid the tubular speculum, which pushes the hemorrhoidal nodes aside, but, under the precaution mentioned in the General Division, use the grooved instrument (page 80). Erosions, ulcerations, proctitis, fissures, fistulæ, etc., must also be looked for.

The *differential diagnosis* is rarely difficult. Broad condylomata can only be mistaken for hemorrhoids when neither has before been seen. It may be more difficult to distinguish hemorrhoids from beginning carcinoma and ulcerations. Since hemorrhoids ulcerate very readily and leave deep lesions, error can only be avoided by careful consideration of all accompanying circumstances. The distinction between rectal polypi and hemorrhoids is readily made, although polypi may closely resemble thrombosed varices. In most instances careful and repeated examination will scarcely leave room for doubt. This is also true of the differentiation between prolapse of hemorrhoids and of the rectum.

TREATMENT

Wherever possible, treatment should be directed toward the underlying cause.

Since constipation is the most frequent etiological factor, we begin with its discussion. In referring to the regulations previously described for the treatment of this condition, we have only to point out special peculiarities which exist in the constipation of these patients. Proper diet is of the greatest importance. We must distinguish, however, between hemorrhoids with and without bleeding, and between insignificant and profuse hemorrhages.

The *diet* must be carefully regulated in patients with severe and habitual or with profuse and periodical hemorrhages. Spiced, sharp, piquant foods and drinks, as well as alcoholic beverages, particularly those of stronger concentration, must be avoided. Further dietetic restrictions are unnecessary. It is obvious that over-action, excessive walking, horseback riding, gymnastics, bicycling, etc., are to be forbidden. If there are no hemorrhages, or if they are insignificant, the diet should be that of chronic constipation, with due consideration to the general health and nutritive condition of the patient. A so-called "bland diet," still advised in many text-books, is *absolutely wrong*. The favourable influences of diet may be further increased by active and passive motion, rowing, room gymnastics, billiard playing, bowling, tennis, Swedish movements, and massage. Owing to the continual local friction and increased circulatory disturbances, horseback riding, and probably also bicycling, act unfavourably.

If diet alone does not produce sufficient evacuations, it must be aided by appropriate laxatives. For this purpose, the sulphur preparations (flowers of sulphur, one teaspoonful t. i. d), and laxatives containing them (compound licorice powder, one teaspoonful morning and evening), have been long and deservedly valued. They operate in accordance with the tenets of the old school: "*Cito, tuto et jucunde.*" Whenever a change of remedies is indicated, the other laxatives mentioned in the chapter on Habitual Constipation may be used, though the above preparations will generally suffice for a long time. The drastic cathartics are said to be harmful in the treatment of this constipation. Recently they have been added to the causative factors of hemorrhoids (Rosenheim [22]). It is certainly an exaggeration to regard drastics as productive of hemorrhoids, but in some instances it does no harm if the physician supports such theories.

Enemata are rarely indicated in hemorrhoids, for manipulations with the usual rectal tubes are apt to irritate, lacerate, and inflame the hemorrhoidal nodules. Chemical agents, including oil, added to enemata, also act harmfully. This applies particularly to glycerin, which causes severe pain and tenesmus.

Almost all text-books advise avoidance of sexual excesses, and claim they may produce hemorrhoids. This statement is true, in so far as these should be avoided by healthy as well as diseased individuals. That sexual excesses are especially harmful to those with hemorrhoids seems to me to be based on mere speculation, and not upon scientific experience. I cannot comprehend how a temporary congestion of the genitals can produce the serious results described.

The use of baths and mineral waters must be briefly touched upon. Only those watering places which contain both appropriate baths and appropriate laxatives (sodium chloride or sulphate) come into consideration. First and foremost are the cold saline springs of Kissingen and Homburg, then the sodium sulphate waters of Marienbad, Tarasp, Elster ("Salt Spring"), Franzenbad ("Salt Spring"), Rohitsch, and others.* In some watering resorts (Elster, Marienbad, Franzenbad) persons anæmic from loss of blood may also use an iron spring. In recent times—even medicine follows fashion, as proved by the yearly increasing number of people who visit these springs—great health-giving properties have been attributed to mineral waters. Convinced by numerous excellent results from a sojourn at these springs, many patients visit them on their own account and recommend their use to others. As physicians, we must as far as possible consider the wishes of our patients, but we should also know the limits of the action of these cures, and not promise greater results than actually occur. We must admit that these springs generally affect the hemorrhoidal disease very favourably. This is quite natural, for the patients find themselves in almost ideal surroundings for the treatment of their disease. The use of aperient waters, the necessary exercise, the appropriate diet, and, not the least, the hygiene of the anal region secured by numerous baths, all combine to produce a condition never, even under the best of circumstances, obtainable at home. But with this, however, balneotherapy finds its limitations. As soon as the patients resume their usual habits of life the old condition returns, and the dearly bought sojourn at the springs loses its magic. It is therefore the duty of the physician to explain to the patient what benefit he may expect from his course of waters, so that gain in health and necessary sacrifice of time and money may be properly proportioned. Permanent good often results from repeated yearly visits to the baths and springs, but this cannot be determined beforehand.

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* [For corresponding springs and wells in the United States, see pp. 161 and 162.—Tr.]

Of the symptomatic remedies for external and mixed hemorrhoids, the first is the *toilet of the anus.* The patient is to keep the anal region absolutely clean. After every act of defecation, this region and the hemorrhoids themselves are to be carefully washed with absorbent cotton (not sponges) dipped in a cold three-per-cent boric-acid solution, or, what I especially recommend, a tannin solution (teaspoonful to a quart of water). Cold antiseptic or astringent washings are very agreeable to the patient, and are to be repeatedly used. We should, however, always study our patients before giving directions. I know from experience that in neurasthenics such regulations may lead to quite unpleasant consequences. The patients examine their anus all day long by means of mirrors and reflectors, just as tongue hypochondriacs do their tongues.

The most important complications of hemorrhoids are severe periodic or marked chronic hemorrhages. In milder bleeding treatment is scarcely necessary. In *severe* hemorrhage it is best not to delay too long with ineffectual remedies such as the introduction of ice, injections of tannin or liquor ferri into the rectum. By means of a speculum we tampon the rectum with gauze or cotton dipped in liquor ferri or ferripyrin solution. As in uterine hemorrhages, hot irrigations (35° to 40° C.) have been recommended by several authors (Sandowski and others). If these measures do not control the bleeding, the bleeding vessels or tissue must be sought for and tied off.*

In *continuous* hemorrhages, particularly where there is anæmia and general weakness, I would recommend witch hazel. I use the fluid extract exclusively in teaspoonful doses three times daily. I have had no experience with the dry extract "hamamelin" (dose, 0.05 to 0.06 gram) recommended by Soulier[23] for the same purpose. Since the different preparations in the market vary in strength, we should use a reliable one. After an extended experience I do not at all doubt the action of hamamelis in hemorrhoidal hemorrhage. The remedy may be taken for weeks and months without producing untoward symptoms. In continuous hemorrhages I have the patients take the drug regularly six to eight weeks.

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* [Firm packing of the bleeding area with dry gauze (if necessary under general anæsthesia) is the simplest and surest of the non-operative means of arresting hemorrhage. The hard clot and the dirty, slowly healing slough after the application of the liquor ferri, make its employment undesirable. I have seen rapid and permanent arrest of rectal hemorrhage follow the direct application of cotton swabs soaked with fifty-per-cent antipyrin solution to the bleeding surface. Perhaps, too, the local use of solutions of suprarenal extract would be effectual. Absolute rest is of course essential to arrest of hemorrhage.—Tr.]

Ergotin, hydrastis canadensis, liq. pot. arsenites, glycerin, and other remedies have been recommended in hemorrhages of this character, but no definite proof of their favourable action exists. Personally, I have had no experience with them.

Reposition is the chief measure in strangulation of varix nodules. This is best carried out with the patient lying on his side, and the hemorrhoidal mass and the parts about freely smeared with an ointment containing cocain, eucain, or opium, or with olive oil. Gentle pressure must be made. Narcosis, when possible, is preferable. Schleich's local anæsthesia, however, is even better. Leeches applied to the anus (but not to the varices) are very useful in reposition. After an abundant hemorrhage the hemorrhoids are easily replaced. When gangrene has occurred the nodules should be dusted with an antiseptic powder (iodoform, airol, xeroform, etc.).*

Besides these important complications, there are inflammatory swellings and excoriations of external and internal hemorrhoids. In the former, anæsthetic suppositories (cocain, eucain, opium, belladonna, and morphin) are generally useful. Unna, Kossobudskji[24], and Macdonald[25] recommend the following suppositories:

℞ Chrysarobin	0.08
Iodoform	0.02
Ext. bellad.	0.01
Butyr. cacao	2.00

D. t. dos. No. x.

S.: Apply one suppository two to three times daily.

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* [If the patient refuse anæsthesia or operation, and reposition otherwise is impossible, rest, local application of ice, and free inunctions with gallic ointment, with opium, cocain, belladonna, etc., are in order. Under these, reduction in size often follows and reposition is then possible. It would be interesting to try the suprarenal extract in these conditions. Occasionally gangrene and a spontaneous cure occur.—TR.]

It would be interesting to know which of these three remedies, chrysarobin, iodoform, or belladonna, is the effectual one.

As may be seen from their composition, styptic properties are attributed to these suppositories. For a like purpose, Rosenheim[*] recommends the injection of a very weak solution of nitrate of silver (one gram of a one-half-per-cent to one-per-cent solution) into the rectum by means of a specially devised syringe. Anæsthetic ointments may also be applied with the collapsable tube, previously described.

In external hemorrhoids the various ointments again come into consideration. Chrysarobin enjoys a special reputation. The ointment recommended by Kossobudskji is as follows:

℞ Chrysarobin	0.8
Iodoform	0.3
Ext. bellad	0.6
Vaselini	15.0

D. S.: To be freely applied several times daily.

We will not enumerate the numerous other salves which probably act only through the anæsthetic drugs they contain. Preismann[26] praises the action of external applications of iodin-glycerin very highly, and gives the following:

℞ Kali iodati	2.00
Iodi puri	0.20
Glycerini	35.00

Later in the disease he increases the strength of the application, thus:

℞ Kali iodati	5.00
Iodi puri	1.00
Glycerini	40.00

Esmarch's rectal support is also to be recommended in prolapsed hemorrhoids (see Fig. 46).

We must distinguish between "bloodless" and "bloody" *surgical measures.* The former include stretching of the sphincter, particularly recommended by French authorities (Verneuil and others). It may be carried out in one or in several sittings by forced dilatation with fenestrated speculum, the blades being separated by a special mechanism. The same result can be more simply accomplished by passing two fingers into the rectum after previous introduction of a grooved speculum and eversion of the rectum. The second bloodless method, used and recommended more especially in England and America, is the fixed or elastic ligature (von Dittel). As far as I know, this procedure is but little used in Germany. Destruction of the nodules with fuming nitric or carbolic acid is also practiced; the skin of the anal region and the perineum is protected from the action of the acid by a thick coating of vaselin.

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[*] Loc. cit., p. 236.

In 1887, Lange[27], of New York, recommended local injections of carbolic acid and glycerin, in concentration of 1:5 to 1:2. In numerous cases, even of large nodules, this method has proved successful in my hands. I usually proceed as follows:

The patient is told to press out the nodules. For this purpose it is best to give him a warm enema beforehand, or to have him sit on a bed-chamber filled with hot water. The rectum and the anus are then carefully cleansed with a one-half-per-cent lysol solution. The skin about the anus is smeared with borated vaselin. As an injection I use fifty-per-cent carbolic-acid glycerin, employing an accurately graduated syringe. The needle is introduced at the border of each nodule, about three drops injected into each, and the needle is allowed to remain in situ for a few minutes. After it has been withdrawn and the parts again cleansed, the other nodules are successively treated in the same manner. If possible, the prolapsed nodules are replaced, a large cotton pad and T-bandage applied, and, to produce constipation, fifteen drops of the tincture of opium and a bland diet given. Rest in bed from two to three days. Castor oil on the third day. Limited activity for several days.

In only one of my cases did acute inflammation follow; under appropriate treatment the inflammation soon disappeared. All the cures resulted without much pain. This operation does not guard against relapses, but it has the great advantages of simplicity and lack of danger.*

The "bloody" methods of operation seek destruction of the hemorrhoids by the production of scar tissue by means of either the actual or thermo-cautery [Paquelin], the galvano-caustic loop (von Bardeleben), or by extirpation of the nodules and subsequent suture

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* Roux[28] very appropriately says of this method: "Compared to the bloody operations it has only one disadvantage: it is no longer an art to rapidly and carefully operate hemorrhoids."

(Whitehead's operation). These are the operations generally performed in Germany; they will be found described in surgical textbooks.

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CHAPTER XXI

NERVOUS DISEASES OF THE INTESTINES

Preliminary Remarks.—In the General Division (page 34) we have given a brief and incomplete description of intestinal innervation. From this it is seen with what complicated conditions we must deal in describing the pathology of intestinal neuroses.

The few anatomico-pathological investigations of Jürgens[1], Blaschko[2], Sasaki[3], Schleimpflug[4], and Emminghaus[5] point the way to future investigators; in themselves they are not sufficient for clinical purposes.

The uncertainty of the study of intestinal neuroses is further increased by the unstable transition between organic intestinal disease and the so-called neuroses. Just as cystitis may develop from vesical paralysis of spinal origin, so organic changes in the intestinal mucous membrane may arise from disturbances of intestinal innervation. At certain stages we can recognise changes, but not their origin. Disturbances of intestinal innervation probably do not follow one course, but secretory-motor and vaso-motor disturbances combine.

Under conditions so little understood, our only resource is clinical observation and experience.

Like gastric neuroses, intestinal neuroses may be divided into motor, sensory, and secretory. There is also a mixed or "complex" form of intestinal neurosis, which appears as general intestinal neurasthenia.

1. Motor Neuroses

(*a*) *Enterospasm and Proctospasm*

While spastic conditions of the bowel are most frequent and prominent in organic intestinal disease (particularly stenosis), enterospasm is very seldom observed as a *purely functional neurosis.*

Nothnagel[6] denies its existence entirely. This seems to me somewhat farfetched, although experienced physicians must admit the rarity of primary, spastic intestinal contraction. Few cases have been reported, and these (including the one of Talma cited by Nothnagel *) are not entirely free from objections. *Proctospasm* is usually secondary to local affections of the rectum or of the pelvic organs. It is met with as a functional condition (occasionally combined with anal crises) in tabes dorsalis, and as a symptom of general hysteria and neurasthenia.

SYMPTOMATOLOGY AND DIAGNOSIS

The principal symptoms of enterospasm are painful intestinal contractions which the patient feels, and which are accompanied by rumbling and borborygmi, and by the passage of fragmentary scybalæ, with marked rectal tenesmus.

These symptoms generally occur at intervals, and are often started or increased by excitement. The following is an example of enterospasm of nervous origin:

Miss H., sixty years of age, has for many years suffered from the following attacks: Every four to eight days, without apparent cause, sudden severe pain is felt in the umbilical region. At the height of the attack there is an urgent desire to defecate. Three to five times a day she passes many small, thin fæcal masses. Each act of defecation is followed by one or two hours of ease.

During these attacks (which often last a whole day) the patient is confined to her room. The abdomen is frequently distended. Except for mild constipation, the patient is subjectively well. She ascribes her affection to overworry. The first attacks began twenty-five years ago. Besides slight hemorrhoids, this very robust and well-nourished woman presents no objective symptoms.

In cases like this the *diagnosis* is readily made, but where the etiology is less clear it may be very difficult to distinguish between this affection and organic stenosis. The following points are important: the long duration of the affection, the good general condition, normal health in the interval between attacks, and the presence of neurasthenic or hysterical stigmata.

The *symptoms* of proctospasm consist of severe periodical, sometimes almost unbearable pain in the rectum, accompanied by sphincteric contraction.

The following cases will illustrate and explain the clinical picture of the disease:

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* Loc. cit., p. 463.

I. (Observation of Peyer[1].) Sexual neurasthenic, aged fifty, suffers from various severe neuralgias, particularly of the testicle. His proctospasm shows itself at first as a marked, painful tenesmus of the rectum, necessitating an immediate attempt at defecation, but neither stool nor wind passes. With this tenesmus there is a very severe spasm of the sphincter, so that the patient cannot force even the narrow canula of an irrigating syringe into his rectum. After some time the pain leaves suddenly and radiates to the bladder or testicle.

II. (Personal observation.) Mr. K., merchant of Prague, aged fifty-two, good family history, had a mild attack of syphilis six years ago. Later, articular rheumatism, which his physician ascribed to the syphilis. Irregular stool during the confinement to bed necessitated by the rheumatism. Though he generally sleeps well, and has a good appetite, he has become very nervous through much overwork. There is constipation, and occasionally mild tenesmus. When tenesmus is severe, diarrhœa ensues; normal, cylindrical stool is very rare.

During the last few years he suffers from anal spasms every two or three days, and more recently every three to five days. These generally come on at night. The patient is suddenly awakened by severe pain, as if the sphincter ani were spasmodically drawing itself together. The finger can be introduced only with great difficulty. The spasm may be controlled by the successive introduction of the larger fingers. In a few minutes the entire attack ceases. The proctospasm is generally preceded by constipation, and never occurs after satisfactory defecation.

In the interval between attacks the calibre of the rectum is normal. No symptoms of tabes.*

The *diagnosis* of proctospasm is not difficult. In order to establish the nervous character of the disease it is always necessary to make a rectal or vaginal examination.

TREATMENT

Where appreciable lesions of the rectum or genitals exist, or where the symptoms are produced by spinal disease, appropriate measures must be applied. In purely functional entero- and proctospasm it will be necessary to treat the underlying nervous basis of the disease. For this purpose cold hydrotherapeutic measures, enemata (perhaps with the rectal cooler), mild galvanization of the rectum and abdomen, and systematic passing of elastic bougies are best. The attack may be checked by bromids, or by opium, morphin, belladonna, codein, and cocain, internally or in suppositories. Constipation, if present, should be treated dietetically, with or without the addition of mild purgatives and enemata.

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* We must always remember the possibility of this being an initial symptom of tabes, which, analogous to gastric crises, may antedate the ataxia by many years.

(*b*) *Peristaltic Restlessness* (*Tormina Intestinorum Nervosa*)

Kussmaul[8], in 1878, was the first to call attention to the occurrence of visible peristalsis of the stomach and intestine in neuropathic individuals. These contractions may affect stomach and intestines together or each separately. They occur in paroxysms, particularly after nervous excitement. They are usually observed in women with flabby abdominal walls and enteroptosis. Peyer[7] has observed several cases in male sexual neurasthenics.

SYMPTOMATOLOGY AND DIAGNOSIS

The chief symptoms are a feeling of movement and of drawing together in the abdomen, which may increase to spasmodic pain. In marked cases there may be simultaneous rumbling sounds. These attacks are independent of the time of food ingestion, but may be visibly increased by certain agents—carbonated beverages, laxatives, etc. They frequently occur at night, and deprive the patient of sleep.

On inspection, the lively peristaltic wavelike motion of the intestine is usually immediately apparent. Where the picture is not well defined, hyperperistalsis may be induced by abdominal friction, pouring ether upon the abdomen, faradism, and distention of the stomach or intestine with air or carbonic acid. The peristalsis mainly involves the small intestine, but, as shown by the case described below, the large intestine may also be affected. Nothnagel[6] maintains that evacuations always accompany the latter condition. In my case this was not so; the patient always had normal stool.

The attacks vary considerably in intensity and duration. In the same individual I have sometimes seen mild peristalsis last for hours, and violent actions decrease after a few minutes.

In my work on Diseases of the Stomach (Part II, third edition, p. 236) I have described a case of this kind. I demonstrated a second much more typical patient at the Congress for Internal Medicine[9]. As the latter case is very interesting I repeat it here:

Pauline R., sixty years old, peasant's wife, has had considerable trouble and worry throughout her life. Her father, a teacher, suffered from nervousness during the last few years of his life, and committed suicide in his fifty-second year. Patient has a brother who is quite well. No other sickness in the family.

Patient herself, when a young girl, was always anæmic, and had "liver trouble," but was never confined to bed, and never had jaundice. Present illness began about eight years ago, when, after passing through a severe illness and after the death of her husband, she spent many weeks in mental suffering. Her symptoms then consisted of *an occasional wavelike motion in the abdomen, which disappeared whenever she felt contented or happy.*

The death of her only son aggravated her condition. Since then (three years) her condition has remained about the same. She is sometimes entirely well for weeks and months, then, generally after some psychic disturbance, she suffers for days or weeks. Since March of this year the symptoms are almost continuous; this she ascribes to the severe illness of her son-in-law.

She complains of a feeling as of a dead weight in the abdomen and a wavelike sensation, which occur usually at night. The attacks are independent of work, and are frequently relieved by eating. Appetite good; bowels regular. Patient does not otherwise feel ill. Has had five children; no miscarriages.

Status præsens.—Frail woman, good colour, poorly developed muscular system and fat. Pupils react well to light, but not so well to accommodation. Triceps and patellar reflexes cannot be elicited. Skin reflexes markedly diminished. Lungs and heart normal. Abdomen is hemispherically distended, the greatest prominence appearing below the umbilicus. When the patient raises the upper portion of her body the intestines press forward through a broad diastasis of the recti muscles, which reaches from the umbilicus to the symphysis. Abdominal walls are thin and flabby.

Liver: Upper border begins at the seventh rib in the mammary line. As determined by palpation and percussion, the lower border of the liver corresponds to a line beginning two fingerbreadths above the right anterior inferior spine of the ilium, passing through the umbilicus, and reaching the eighth left costal cartilage. Above this line there is uniform dulness. The sharp border of the liver may be plainly felt in the right half of this line, less so in the left half. The spleen is slightly movable, and its border may be felt in the left hypochondrium.

Kidneys are palpable.

As determined by palpation of an introduced sound, distention, and transillumination in the fasting condition, the greater curvature of the stomach extends a handbreadth below the umbilicus. During fasting the stomach is empty. Test breakfast shows normal gastric functions. The urine contains no albumin, sugar, or indican. Microscopical and macroscopical examination of the stools shows no changes.

Through the thin, flabby abdominal walls one can plainly see the peristaltic movements (Fig. 47). Three types may be distinguished; they vary in position, form, and course.

During rest, one sees two or three parallel sausage-shaped protuberances lying quite closely together. These periods of rest last but a very short time. Soon each protuberance is seen to contract at one end. The wave of contraction passes along the entire segment, while behind it the protuberance again reforms. The whole action is slow and vermicular. There is a continuous alternation of contraction and protuberance, and, following the simile of Nothnagel, the surface of the abdomen may be compared to that of a bag filled with potatoes.

In the left side of the abdomen, from the free border of the ribs to about the anterior superior iliac spine, five—occasionally six—parallel swellings, each about the thickness of a stout lead pencil, may be seen. They are somewhat farther apart than the sausage-shaped tumours before described, and approximately one half the length of the latter (about 4 to 5 centimetres). Several of these swellings disappear and reappear one after the other, thus giving the impression as of the whole mass suddenly springing forward ("harmonica motion"). Seemingly independent of the motions just described, a line of shadow passes about midway between these tumours, oscillating from left to right and *vice versa*.

In the epigastrium a semicircular arching may be seen, the lower border corresponding exactly to the greater curvature of the stomach. Along this border a deep contraction passes from left to right, and is immediately succeeded by a protrusion. One or two fingerbreadths below the liver the contraction continues somewhat longer, so that we see two protrusions separated by a depression; from this point the contraction again takes place more energetically, till the line of liver dulness is reached. The series then begins anew and continues uninterruptedly. The direction of motion is always from left to right. Each series lasts from fifteen to eighteen seconds.

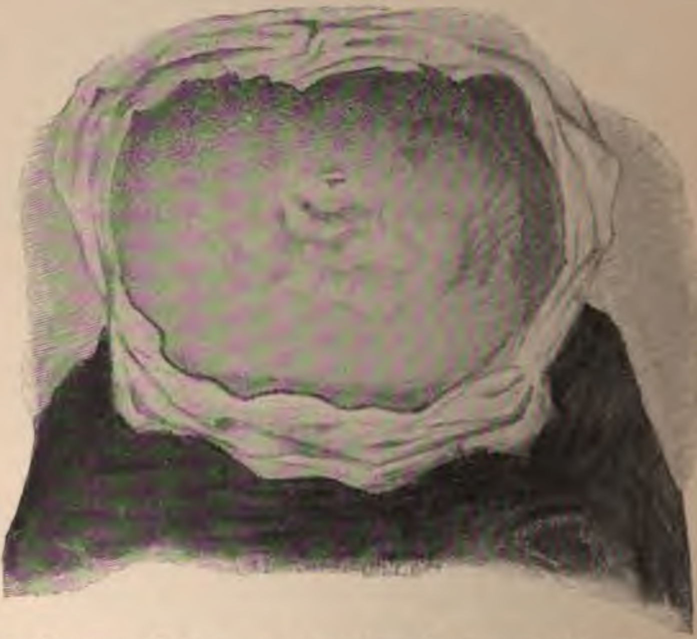

FIG. 47.—PERISTALTIC RESTLESSNESS OF THE SMALL INTESTINES AND DESCENDING COLON. (Original observation.)

The patient does not feel these motions. Throughout the examinations she has pain only when the peristalsis becomes severe. A pulsation can also be seen; it may be mistaken for peristalsis. The pulsation evidently originates in the great arterial trunks, and is transmitted through the flabby abdominal walls. It can be seen, and not felt, and is synchronous with the radial pulse.

These peristaltic movements may be increased to a slight extent by faradization, friction, and cooling the abdomen.

The *diagnosis* of the condition is not difficult. We must always consider the possibility of intestinal stenosis or partial adhesions. Several years ago I observed a large, fixed umbilical hernia complicated by hyperperistalsis. The condition of the general nervous system, the alternate disappearance and reappearance of the peristaltic movements, and its dependence upon excitement, are useful diagnostic facts.

TREATMENT

We must first attempt to strengthen the general nervous system through appropriate climatic and hydrotherapeutic measures. Kussmaul has achieved good results from the intragastric and external use of the faradic current.

Internally, we may give the alkaline bromid salts, narcotics, and perhaps also antipyretics (antipyrin, phenacetin, lactophenin, etc.). In one case I saw good results from codein and belladonna. Rosenheim[10] recommends chloral hydrate (1 gram, evenings, in gruel).

In the case described the attacks ceased when the patient was admitted to my private clinic for closer observation. Bodily and mental rest and appropriate nursing evidently contributed considerably to this favourable result.

(*c*) *Paresis* (*Atony*) *and Paralysis of the Intestines*

Paresis or atony of the intestines ("intestinal insufficiency," O. Rosenbach) is a functional debility of the intestinal muscular system; paralysis is an absolute loss of the motor power of the intestines. Both conditions affect the large bowel only. Pareses of the muscle of the small bowel are unknown. In the chapter on Intestinal Strictures and Intestinal Obstruction we have described paralytic conditions of mechanical origin. In what follows we shall discuss only their functional phases.

(1) ATONY OF THE LARGE INTESTINE

This is closely related to and as frequent as constipation. All the factors which produce constipation may in time produce flabbiness or muscular fatigue of the bowel, and, as a result, partial or general distention, or even actual dilatation of the intestine. Constipation is here the primary factor. Much more difficult is the determination of other conditions which produce primary paresis of the nervous muscular apparatus. By the previously described experiments of Emminghaus, it was shown for the first time that degenerative processes of the splanchnic nerve may produce constipation; this constipation must be considered as an example of neuropathic intestinal atony. The artificial or toxic intestinal pareses following the use of opium, morphin, and belladonna must be placed in the same category. In a case under my own observation, a woman who within a few weeks had taken more than one kilogram [2.2 pounds] of bismuth for symptoms resembling gastric ulcer, there developed most marked intestinal paresis and dilatation. It is well known that individuals who suffer from neuroses and psychic disturbances (neurasthenia, melancholia, hysteria, hypochondria, etc.) frequently develop paretic or subparetic conditions of the intestinal muscular apparatus. Thus, in persons predisposed to habitual constipation, I have occasionally observed acute intestinal paralyses from sudden fear, anger, or other excitement. We may explain these phenomena by reflex irritation of the nerves that inhibit intestinal peristalsis (splanchnics). In view of analogous conditions in the stomach, we must also admit that traumatism and shock may produce an inhibitory action upon the motor apparatus of the large intestine. Congenital atony of the intestine has also been observed. Intestinal paresis may result from the continuous use of large rectal enemata.

Clinical observation has led to the differentiation between general and partial atony. In a series of articles Federn[11] has called attention to the latter condition. According to this author, partial atony plays a large part in general pathology. He finds this condition not alone in intestinal diseases, but also in arterio-sclerosis, cardiac asthma, pulmonary tuberculosis, neurasthenia and hysteria, and Basedow's disease. Federn even states that partial intestinal atony is connected with the development of the last-named disease. He maintains that the diagnosis of partial atony can be made from the strikingly pungent smell of the stools, and from the fact that gentle percussion of the intestines elicits dulness over some areas, while deep percussion of the same areas elicits a tympanitic percussion note.

It needs no special demonstration to prove how little defined and characteristic are the symptoms described by Federn. Nevertheless, there is a grain of truth in Federn's observations, a truth of which experienced physicians have not been as ignorant as this author would have us believe. At several places along the intestines, as the result of fæcal stagnation, partial ballooning may develop, and despite the presence of diarrhœa (stercoraceous diarrhœas), fæcal stagnation may exist. As is well known, the sites of predilection for fæcal accumulations are the cæcum, the hepatic and splenic flexures, the sigmoid flexure, and the ampulla of the rectum.

M. Herz[12] has recently directed attention to a further type of intestinal insufficiency—that of the ileo-cæcal valve. Normally, the large intestine is shut off from the ileum by this valve. From inflammation or by flattening of its lower fold there may result a relative insufficiency of the valve, as shown by tympanites, constipation, flatulency, and neurasthenic and other nervous symptoms.

Symptomatology and Diagnosis

These have in greater part been described in the chapters on Habitual Constipation and Chronic Enteritis. Atony is frequently associated with the latter condition.

Atony is most easily demonstrated by the method described in the General Division of this work (page 74). It consists in the injection of measured quantities of water per rectum and the eliciting of splashing sounds in the corresponding segments of the large intestine. Upon distending with air, we find that the amount required is far above the normal; this also speaks for abnormal flabbiness of the intestine. Herz believes that proof of ileo-cæcal insufficiency is demonstrated when gas under pressure can be forced from the cæcum into the ileum. The changes in the percussion note are best obtained by percussing with the edge of the fingernail of one hand upon the nail surface of the other.

The clinical aspect of diffuse atony is so characteristic, that its *diagnosis* will rarely be difficult. In every case, however, we should think of the possibility of an underlying mechanical cause.

As long as we have no more definite data than that obtained from percussion, we shall not be able to diagnose partial intestinal atony.

Treatment

This is identical with that described under Habitual Constipation and Chronic Enteritis (*q. v.*). It is unnecessary to repeat the detailed regulations there given. In ileo-cæcal insufficiency, Herz recommends massage of the large bowel. In partial atony, besides massage, Federn advises faradization of the large intestine.

(2) PARESIS AND PARALYSIS OF THE RECTUM

In describing paralytic ileus (page 403) we also discussed paralysis of the large intestine. It still remains to describe paresis of the rectum.

Chronic paresis of the rectum generally results from local rectal affections (prolapse, tumours, proctitis, hemorrhoids, etc.), but occurs also as a symptom of some spinal and cerebral lesions (locomotor ataxia, progressive paralysis, myelitis, etc.). Straining during defecation and urination in prostatic hypertrophy and stricture of the urethra may likewise produce rectal paralysis. As a purely neurotic affection this condition is extremely rare. I have once seen it in a boy of nine years who was convalescing from diphtheria.

Several degrees of rectal paralysis are met with. In mild cases the rectum is only relatively incontinent; in the severe it is absolutely so.

SYMPTOMATOLOGY AND DIAGNOSIS

The main symptom is loss of voluntary control of evacuations. In mild cases the sphincter is incontinent only when there is diarrhœa; in the severe, formed stools are also involuntarily passed. Active movements of the body, slight straining of the abdominal muscles, coughing, laughing, and sneezing may cause involuntary evacuations and thus distress the patient.

The *diagnosis* can at once be made by digital examination of the rectum. The question of etiology is more difficult. We must search for not only disease of the rectum and of intestinal segments higher up, but also for spinal disease. As shown by the following history, it may not be easy to arrive at the proper explanation of some of these cases.

F. S., aged twenty-two years, book gilder, has lost control over his stools for the last two years. As soon as there is a desire to defecate, no matter whether the stools be solid or fluid, an involuntary evacuation immediately follows. The patient also complains about urination; he must wait some little time before the urine begins to flow. The act of urination is normal and does not indicate stricture. Denies gonorrhœa and other sexual diseases.

Patient works considerably with metallic dust which does not contain lead.

Status Præsens.—Healthy-looking young man; internal organs normal. Anus very flabby, readily admitting two fingers. Proctoscopy shows nothing special. Patellar reflexes markedly increased; no disturbance of sensibility.

The diagnosis of beginning spinal lesion was made. This was concurred in by Professor Oppenheim, who, in view of the intact sensibility, diagnosticated a lesion in the motor centres of the bladder and rectum.

TREATMENT

Where the rectal paresis is secondary, treatment must be directed toward the underlying cause. Where this is impossible, or where a primary neurosis is present, we should endeavour to improve the functions of the incompetent sphincter. For this purpose, faradization of the rectum is of the first importance, and must be carried out thoroughly and systematically. The diet should vary with the degree of the paralysis.

If involuntary evacuations occur only when there is diarrhœa, the latter must be prevented by suitable dietetic regulations (astringent diet). If involuntary evacuations occur when the stools are well formed, we must prevent fæcal accumulation in the lower intestinal segment. By diet we should attempt to have the stools semisolid, so that a complete daily evacuation follows. The patient must be particularly impressed with the importance of attending to defecation regularly, at a certain fixed time of the day.

Where, despite these precautions, fæces accumulate in the rectum, they should be got rid of by enemata of oil or soap water. The diseased region should also be stimulated by cold sitz baths and frequent irrigations. Of medicinal remedies, injections of strychnin (0.001 to 0.002 gram per dose), or suppositories of nux vomica (0.03 gram twice a day) are best.

(*d*) *Nervous Flatulence*

This consists of alternating expulsion from and reaccumulation of air within the bowel, similar to nervous eructation. It is seen mostly in hysterical girls and women and in neurasthenics, but also occurs in healthy individuals.

The affection may be acute, chronic, or periodical. As yet it is but little understood. The best theory is that which attributes it to rhythmical contraction and dilatation of the intestine. Analogous to what Oser believes to take place in nervous eructation, after the air has been expelled from the lower bowel, renewed contractions draw more air from the upper segments; this leads to dilatation, and when the bowel is quite distended the air is expressed, etc. In well-marked cases the expressed air is odourless, or only slightly mixed with offensive gases.

Symptomatology and Diagnosis

A feeling of tension and pressure predominates; this may increase to severe colicky pain. Occasionally the air is heard rumbling through the gut. The abdomen may be more or less distended; in my cases, however, despite repeated complaints of the accumulation and passing of air, abdominal distention was scarcely appreciable. Passing of wind affords but little relief. Gases reaccumulate, and the patients are disturbed throughout the day and sometimes at night. The symptoms are aggravated by mental disturbances.

The *diagnosis* is made from the clinical phenomena, the course of the affection, and the negative abdominal symptoms.

Treatment

This should be directed toward the underlying hysterical or neurasthenic basis. The forbidding of foods which produce flatulency is usually ineffectual. Since the patients are generally poorly nourished, anæmic individuals, a mixed diet rich in fats is especially beneficial. Fluids and soups should be avoided, since they often increase the flatulence and the feeling of weight in the stomach and intestine. Warmth, both internal (valerian, peppermint, and caraway teas) and external (warm fomentations), brings relief and quiets the excited peristalsis. Of the many remedies recommended for nervous flatulence, the best are the nux vomica preparations (extract of nux vomica, 0.01 to 0.03 gram per dose, in powder or pills), or extract of Calabar bean (0.05 gram to 10.00 of glycerin, 5 to 6 drops t. i. d., or pills, 0.005 to 0.01 gram per dose).

Where constipation coexists, diet and the magnesia preparations (especially magnesia usta) are to be recommended.

2. Sensory Neuroses

Enteralgia (Neuralgia Plexus Mesenterici)

This is a periodic, painful irritation of the intestinal nerves, occurring without apparent anatomical cause. Thus enteralgia is a true neuralgia, and, as stated by Nothnagel,* must be distinguished from the colic of intestinal contraction. The cause is said to be in the large nerve plexuses (mesenteric, hypogastric, and cœliac).

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* Loc. cit., p. 489.

Romberg has tried to establish separate types of the affection for the different plexuses affected. When carefully examined, however, the symptoms are so similar that it is almost impossible to differentiate these groups. The very assumption that enteralgia is due to disease of the mesenteric plexus is purely theoretical. Malaria, lead poisoning, and locomotor ataxia are cited as causes of enteralgia. This condition is also found in hysterical and neurasthenic individuals, and Peyer has very often observed it in sexual neurasthenics.

SYMPTOMATOLOGY

Enteralgia presents itself as mild or severe, drawing, cutting, boring, and burning pains in the abdomen, particularly in the mesogastrium. The patients take to bed, writhe with pain, and seek relief by evacuations or the passing of wind. In severe cases vomiting may occur and afford the patient temporary relief.

We shall not discuss in detail the enteric crises of locomotor ataxia, but mention that they constitute the purest and most instructive types of enteralgia.

The attacks may cease after several minutes, or, with slight remissions, last for hours or days (as in visceral crises). They generally cease or become less acute during the night.

In one of my patients, a man of fifty, whose nervous system was upset by intense excitement, severe pain followed each act of an otherwise normal defecation. Careful clinical examination showed normal intestinal functions and stools.

Galvanism improved his condition, and a sojourn in the mountains entirely cured him.

Objectively, there may be slight tympanitis and local points of pain, particularly in both hypochondria (A. Peyer). According to my experience, the severe pain complained of is in marked contrast with the normal condition of the abdomen.

Constipation may accompany enteralgia, but, unlike stercoraceous colic, does not constitute a necessary feature of the attack. That constipation accompanies enteralgia of longer duration (e. g., enteric crises) is explained by the vomiting usually present, and the abstinence from food.

The *diagnosis* of enteralgia is easily made when its etiology is apparent (e. g., locomotor ataxia, arthritis, lead intoxication, hysteria, and neurasthenia). When the etiology cannot be discovered the diagnosis is always uncertain.

Hepatic, renal, and cystic calculi must first be carefully excluded. An irregular cholelithiasis may closely resemble enteralgia. Careful examinations of the anterior and posterior regions of the liver, questioning regarding icterus, and the finding of concretions may aid in the differentiation. In distinguishing from nephrolithiasis and cystolithiasis, we must consider the data obtained from palpation of the kidney and bladder, examination of the urine, and cystoscopy. Enteralgia can usually be distinguished from flatulent colic by the presence of fæcal accumulations, or by the irregular character of the stools in the latter and by the cessation of the attack when large quantities of flatus have been passed.

Differentiation from true colitis can generally be made by repeated and careful inspection of the stools and by intestinal irrigation. In this connection we would mention herniæ of the linea alba, which are easily overlooked, and which often cause gastralgia or enteralgia. Adhesions of the intestinal segments may produce all the symptoms of severe intestinal neuralgia.

In some text-books (Rosenheim and A. Pick) peritonitis and perityphlitis (A. Peyer) are also considered in this connection. When the patient is examined not only during the attack but also after its completion, error is almost impossible.

TREATMENT

As enteralgia is not a primary affection, its curability depends upon the underlying disease. In lead colic, complete cure is accomplished by removal of the toxic cause and by the administration of appropriate remedies (iodin preparations, sulphur baths, etc.). Up to the present time the visceral crises of locomotor ataxia have remained rebellious to treatment. The crises of gout are likewise extremely obstinate. In these latter instances, as well as in idiopathic enteralgia, we must treat the paroxysms symptomatically with hot fomentations, warm enemata, and narcotics (best subcutaneously or in suppositories). Invigorating general treatment is indicated in neurasthenical or hysterical enteralgias. Galvanization of the abdomen and rectum may also be tried.

3. Secretory Neuroses

(*a*) *Nervous Diarrhœa*

This condition, first minutely described by Trousseau, is characterized by more or less numerous thin, watery, generally periodic evacuations. The diarrhœa may result from some central disturbance (the irritation being carried along the course of the vagi and sympathetic), from peripheral irritation (alimentary diarrhœa), it may be reflex (disease or displacement of the genital organs, entozoa, thermic causes, etc.), or may be due to the absorption of toxic products.

In a limited sense, nervous diarrhœa is usually an accompanying symptom of general nervous debility. This fact must be kept in mind, since only by its careful consideration can a positive diagnosis be made and favourable treatment instituted. A more extended experience enables us to distinguish several types: The first group is characterized by the fact that under the influence of emotions, or after partaking of certain food or drink, these individuals, whose intestinal functions are otherwise normal, suddenly have one or several quickly repeated fluid evacuations.

In the second group the intestines are unstable, a tendency to diarrhœa exists, but the patients are otherwise healthy. From mental excitement or variations from their ordinary mode of living, sudden severe diarrhœal evacuations occur, which cease after the patient is placed under normal conditions.

A third group is characterized by diarrhœa under special conditions—for example, when opportunity for defecation does not exist, or is surrounded by certain embarrassing difficulties. At other times the intestinal function is normal.

Symptomatology and Diagnosis

Apart from the etiology and the periodicity of its occurrence, nervous diarrhœa presents no specific features. The number of dejections varies largely. The subjective symptoms are the usual ones of tenesmus, rumbling, severe thirst, etc. The stools are not characteristic. The patients tell us that food has been passed completely undigested, but since there is rarely an opportunity for examining the stools during an attack this statement must be accepted with great reserve. We shall again refer to the question of mucous admixture of the evacuations. Blood is never or very exceptionally present. Pus is always absent.

These diarrhœas are, so to speak, explosive in character. A feeling of intestinal quietude very soon follows an attack developed with inconceivable rapidity, and then, aside from a slight weariness, the patient is relieved and well. In other cases the attack begins very suddenly, but if left untreated it may continue many hours or days. I have recently seen a very typical case of this kind.

The etiological factors of the attacks vary, and depend chiefly upon the particular psychic idiosyncrasy of the individual. In an excellent article on Gastric Neuroses, Fleiner[13] relates how students before their first duels, and physicians before applying forceps during labour, previously visit the water closet.* These are examples of very acute emotional diarrhœa (diarrhœa produced by fear). In chronic cases, occupation frequently determines the attacks. For example, I have often observed nervous disorders in bankers and stock speculators under the influence of sudden great financial crises. In actors, a début, a gala or a first performance may induce peristaltic hypermotility. Canstatt[14] reports the case of a physician who was attacked with watery diarrhœa before every large operation. Numerous other examples might be given.

Before proceeding to the diagnosis I shall describe several cases which illustrate the above remarks.

I.—Mr. S., wine merchant of Berlin, forty-six years of age; comes from a nervous family, and, as he himself says, has been nervous for years. Was formerly addicted to excessive drinking, especially of champagne. Patient is married to a very nervous and jealous woman. He complains of the following attacks which occur from three to five times yearly: After any great excitement he is suddenly attacked by severe gastric colic, together with profuse diarrhœa. Even the blandest nourishment is passed in a few minutes. The attacks last three or four days, during which time the appetite is very poor. The patient improves gradually. In the interval between the attacks the gastric functions are normal.

The last attack occurred on June 3, 1898, in consequence of great agitation at his home. The patient suffered intense pain, causing him to toss about and to break out into a cold perspiration. The pain always begins during the day, but ceases at night. It is independent of food ingestion. There are twenty to thirty almost watery stools per day, accompanied by severe tenesmus.

Status Præsens.—Muscular, florid-looking man; pulmonary organs normal. Marked diffuse sensitiveness to pressure in the gastric region, disappearing when the patient's attention is withdrawn. Posteriorly, alongside the vertebral column there are numerous scattered pressure points. Patellar and pupil reflexes are somewhat sluggish. No disturbances of sensibility, no Romberg's symptom.

This case gives the impression of a visceral crisis, and we cannot exclude the possibility that the above symptoms represent beginning locomotor ataxia. At present, however, corroborative symptoms are absent, so that we may regard the case as one of secretory neurosis. The etiology also speaks in favour of this diagnosis.

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* In his celebrated novel Débacles, Zola has forcibly described the reflex action upon intestinal peristalsis resulting from the enemy's artillery fire.

II.—Mrs. L. T., of Berlin, aged thirty-three; teacher. For years patient has suffered from nervous disturbances of the stomach and intestines; for example, vomiting and diarrhœa occurred frequently at examinations. Later, this condition improved, but about three months after an operation for fistula in ano it returned. Since that time diarrhœa frequently occurs after the slightest excitement; thus the thought of consulting a physician or of meeting strangers produces intestinal hyperperistalsis. There is also a predisposition to diarrhœa when the patient makes a social call and cannot reach the toilet room unobserved. In her own home, however, such attacks do not occur. Diet has absolutely no effect upon the attacks—e. g., the stools may be quite normal after eating fruit, vegetables, rye bread, or cake. Appetite always good. Patient is anæmic, has cold extremities (*mains serpentines*). Except for mild sphincteric paresis all organs are normal.

Treatment.—Arsenic, which, according to patient, is very successful.

III.—Mr. L., fifty years old, director of a chemical factory in Boston; antecedents of both parents nervous. The sisters of the patient are more or less neurotic, but severe diseases of the nervous system have never appeared in the family. The patient himself is easily excitable, but is of happy temperament.

Has had his present trouble for ten years. It shows itself in occasional diarrhœa, which occurs particularly when the patient is prevented from or embarrassed in seeking the toilet. This is specially the case during railroad trips. He must always ride in a railroad coach containing a toilet. When the patient is invited to dinner it often happens that he must leave because of sudden intestinal hypermotility. When going to the theatre he always takes an end seat, so as to be able to leave quickly if necessary. Otherwise the stools are entirely regular; the appetite is fair; sleep and general condition good.

The patient is a strong, well-nourished man. Objective signs (including examination of the fæces) are normal.

IV.—Mrs. G., of Moscow, aged twenty-nine; merchant's wife. The present affection dates back six years, and began with her puerperium. It commenced with severe intestinal colic and rumbling and fluid stools mixed with mucus and blood. Improvement after three or four days. Since then there has been marked sensitiveness of the intestines, particularly when the patient is excited. For example, the colic and diarrhœa begin when she is frightened or worried; once an attack occurred when her child was sick, and again when she had toothache and migraine. Rest has a favourable effect. Diet has no appreciable influence. In the intervals between the paroxysms the patient has normal stool daily or every second day. During pregnancy and after labour the attacks are more severe and frequent.

On June 19, 1898, the patient was admitted to my private clinic for careful observation.

I pass over unimportant details and mention only the intestinal phenomena.

There is marked sensitiveness along the entire cour
particularly over the cæcum, the descending colon, anc

Examination of the Fæces.—These are formed and
cious mucus. Microscopically there is much degenerat
places striated ground substance, in which, by the
numerous nuclei are seen. Test lavage of the intestine a
or gelatinous masses of mucus.

Treatment.—Astringent diet as in chronic diarrhc
June 25th, after a long visit of a relative from Leipzig,
ations accompanied by severe pain. Despite fluid di
tenesmus and about twenty movements during the ni
stools were brown and watery; microscopically they
Tincture of opium and warm fomentations were orde
feeling of marked tension in the abdomen and of great
soon became firm and less frequent as the patient ra
the clinic on July 5th. After a course at Franzens
cured.

The *diagnosis* of nervous diarrhœa may b
Where, as in the second and third cases, the
dominant, the affection occurs periodically wit
during the intervals, the diet has no influence u
disease, the clinical examination shows other s
thenia, and the stools exhibit no signs of ca
diagnosis can readily be made.

If, however, the condition becomes chronic,
simple. Thus, in the fourth case there is as
against the assumption of a nervous origin. T
in the dejections—a point on which Nothnagel
according to my own experience and that of
no means a positive differential fact. Despite
the disease, a catarrh may develop, or an otl
enteritis may, from mental excitement, suddenl
present symptoms of a severe intestinal cata
mucus in the stools, and yet in both instanc
neurotic.

In his excellent treatise von Engelhardt a
other facts for the differentiation between true
tinal neuroses. He distinguishes between the
follows:

Chronic Intestinal Catarrh.—There is gen
and anæmia. When diarrhœa takes place it i
night or in the early morning, and comes on a
during the day. Diet has a marked effect u

the stool and upon the patient's general condition. There is sensitiveness to pressure over the colon.

Intestinal Neuroses.—No loss of weight; frequently robust appearance despite diarrhœa for many years. The diarrhœa generally occurs at the usual time of defecation or immediately after eating. Evacuations follow one another rapidly, and then cease for a long time. Diet has almost no effect upon the attacks, or they may cease after a mixed diet. Sensitiveness to pressure over the aorta and the iliac arteries.

The facts cited by von Engelhardt are doubtless of diagnostic value, as the clinical histories just given illustrate. In individual cases, however, his points of differentiation may leave us in the dark. For example, I have repeatedly seen morning diarrhœas in typical neurasthenics whose intestinal functions were otherwise normal. Again, the general condition of a patient with intestinal neurosis is not, as von Engelhardt maintains, always excellent. In the second case I have described I find in my journal "the patient lost 5 kilograms within a short time." It follows, therefore, that in view of the innumerable possible combinations of the protean picture of intestinal neurosis, all scientific considerations may occasionally mislead. A clear conception of the disease is possible only from systematic and most careful observation; often we can only establish the diagnosis from the course of the affection, or from the results of treatment.

TREATMENT

This will vary with the type of the disease. The classification previously made (page 535) shows that in the intervals between attacks the intestinal function is sometimes normal. In such cases treatment will be directed toward the general condition, the general and local neurotic irritability, and the diarrhœas themselves when these become excessive. For the last-named condition opium is undoubtedly the most appropriate remedy. Dietetic treatment is of secondary importance. In continuous intestinal irritability the treatment should be different. A diet similar to that of chronic diarrhœa (see pages 224 and 225) will frequently, though not always, prevent an attack. In chronic neurotic diarrhœa the same principles apply. In addition, hydrotherapeutics, and in some cases electricity (galvanism), may be beneficial. For obvious reasons the best results are achieved by sanitarium treatment.

Of medicinal remedies, the bromid preparations (bromid of soda, 0.5, one powder t. i. d.) deserve first consideration. According to Nothnagel (see also Case II), the arsenical preparations (Fowler's solution, 3 to 5 drops t. i. d., in peppermint water) may have a favourable effect. For anæmic patients, iron springs (Franzensbad, Elster, Pyrmont, Cudowa, Rippoldsau) may be of benefit. Carlsbad thermal water taken hot and in small doses is sometimes followed by good results.*

(*b*) *Mucous Colic*

In discussing membranous enteritis (page 228) we mentioned mucous colic and the different theories regarding its origin. There can be no doubt that periodical membranous dejections are frequently observed in neurasthenic and hysterical women. But—and this is the distinguishing characteristic—they scarcely ever occur without simultaneous habitual (generally spastic) constipation. Since we know that in predisposed individuals conditions of obstinate constipation form the basis for all kinds of nervous and hysterical symptoms, we do not consider mucous colic as a symptom of hysteria or neurasthenia, but only of constipation. When the latter is well marked mucous colic occurs, and it ceases when the constipation is no longer present. I do not know of a single instance, either from personal observation or from literature, in which the mucoid dejections did not cease when the bowels were regular. In view of these facts, the theory still maintained by several authors that mucous colic is a secretory neurosis is incorrect.

It is therefore unnecessary for us to give a separate description of this affection; that in the section on Membranous Enteritis suffices.

4. Complex Intestinal Neuroses

Intestinal Neurasthenia

We have stated (page 521) that abnormal disturbances of function may frequently find their outlet along the course of different nerves, so that motor, sensory, secretory, and probably also vasomotor disturbances of innervation may be variously combined. In this manner there result intestinal conditions similar to those produced in the stomach by nervous dyspepsia.

Cherchewski[16] was the first to give a detailed description of intestinal neurasthenia. He directed special attention to three characteristic symptoms, viz.:

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* [For corresponding springs and wells in the United States, see pp. 161, 164, and 165.—Tr.]

1. Habitual constipation, rarely alternating with diarrhœa.
2. Abdominal distention, particularly in the region of the false ribs.
3. Loud, tasteless, odourless eructations which only exceptionally are acid.

These symptoms are also found in spastic constipation, in nervous flatulence, and in membranous enteritis. (Instances of the latter condition doubtless figure among the author's cases.)

Under the term "nervous digestive weakness," Möbius[17] some years ago described a widely different type. In this, despite excellent appetite and abundant nourishment, the patients, without suffering any subjective digestive symptoms, become more and more emaciated. Their evacuations are apparently normal, but really are overabundant, and a large part of their nourishment undoubtedly passes away unabsorbed.

It is well known that, notwithstanding sufficient nourishment, individuals may persistently emaciate, but it is questionable whether we are not dealing with minute, as yet unknown, anomalies of food metabolism. In my opinion, there is at present no reason for a special description of intestinal neurasthenia as an individual clinical picture such as is found in the text-books of Rosenheim and Pick. If we possessed a more or less complete clinical syndrome, we would be justified in separating this from the other forms of intestinal neurosis. Since such is not the case, we must limit ourselves to the statement that functional disturbances of the most varied kinds may be combined with one another, and thus produce an *ensemble* whose individual traits are entirely dissimilar.

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